

Guide to Oral Pathology

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Guide to Oral Pathology

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Guide to Oral Pathology

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Dedicated to

*My parents for their
untiring support without which
I could not have pursued
Dentistry at all*

Preface

This book is named as guide instead of a textbook since the focus of this book is the syllabus of the undergraduate students, and the orientation is towards examination. The entire faculty of Department of Oral Pathology at Sree Balaji Dental College and Hospital was involved in bringing out this guide. But for the spade work done by the postgraduate students Dr JP Rajguru and Dr SB Patil this book would not have been possible.

Since this guide is basically a compilation work from different reference books of international repute, there is no claim by the authors as to the originality of the text. The language is kept very simple and only the most popular concepts and theories have been presented. Histopathology diagrams are given only for the common lesions which we believe an undergraduate student must know.

If there are inaccuracies and ambiguities found anywhere in this book, your suggestions and feedback are welcome at e-mail:masthanmk@yahoo.com

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1

Developmental Disturbances of Oral and Paraoral Structures

DEVELOPMENTAL DISTURBANCES OF THE JAWS

Developmental disturbances are diverse group of deformities in the embryonic development of the head and facial structures. Anomaly as a medical term which means irregularity or deviation from normal. These abnormalities may be present as congenital (Present at birth) or inherited (transmitted genetically). Developmental disturbances may be mild or severe. Numerous factors contribute to their development.

AGNATHIA

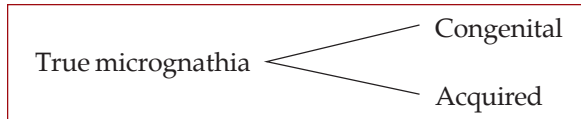
(Otocephaly, Holoprosencephaly Agnathia)

- Extremely rare congenital defect characterized by absence of the maxilla or mandible.
- Only a portion of jaw is missing.
- In maxilla there may be one maxillary process or even the premaxilla is involved.
- In mandible the entire mandible of one side may be missing, or only the condyle or entire ramus will be missing.
- There may be bilateral agenesis of the condyles and of the rami.

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MICROGNATHIA

- Means a small jaw.
- Either the maxilla or mandible may be affected.



- Associated with congenital heart disease and Pierre Robin syndrome.
- Micrognathia of the maxilla is frequently due to a deficiency in the premaxillary area and patients with this deformity appear to have middle third of face retracted.
- Micrognathia may be one of the predisposing factors in mouth breathing owing to associated with maldevelopment of the nasal and nasopharyngeal structures.
- Acquired type of micrognathia is of postnatal origin and usually results from a disturbance in the area of the temporomandibular joint.

MACROGNATHIA

- Refers to the condition of abnormally large jaws.
 - Increase in size of both jaws is frequently proportional to a generalized increase in size of the captive skeleton.
 - General factors which conceivably influence and find to favor mandibular prognathism are.
 - Increased height of the ramus.
 - Increased mandibular body length.
 - Increased gonial angle.
 - Anterior positioning of the glenoid fossa.
 - Decreased maxillary length.
 - Prominent chin button.
 - Varying soft tissue contours.
- e.g. Pituitary gigantism, Paget's disease leontiasis osseae, acromegaly.

FACIAL HEMIHYPERTROPHY

It is a rare developmental anomaly characterized by asymmetric overgrowth of one or more body parts.

More commonly known as hemihypertrophy.

Can be an isolated condition, but also may be associated with a variety of malformation syndromes.

Malformation syndromes associated with hemihyperplasia:

- Beckwith-Wiedemann syndrome
- Neurofibromatosis syndrome
- Klippel-Trenaunay-Weber syndrome
- Proteus syndrome
- McCune-Albright syndrome
- Epidermal nevus syndrome
- Triploid/diploid mixoploidy
- Langer-Giedion syndrome
- Multiple exostoses syndrome
- Maffucci's syndrome
- Ollier syndrome
- Segmental odontomaxillary dysplasia

Classification

- Complex hemihyperplasia
- Simple hemihyperplasia
- Hemihyperplasia involving one side of the face.

Etiology

Unknown. May be vascular or lymphatic abnormalities, CNS disturbances and chromosomal abnormalities.

Clinical Features

Enlargement of face confined to one side of the body. Unilateral macroglossia, premature development, eruption, increased size of the dentition.

Familial occurrence is also seen.

Oral Manifestations

Alteration in the crown size, root size and shape and rate of development.

May affect both dentition.

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Most commonly cuspids, premolars and first molars are involved. Bone of the maxilla and mandible are enlarged. Sometimes altered trabecular pattern.

Buccal mucosa appears velvety, seem to hang in soft, pendulous folds on the affected side.

● **Treatment and Prognosis**

No specific treatment.

FACIAL HEMIATROPHY

(Parry Romberg syndrome, Romberg Parry syndrome, progressive facial hemiatrophy, progressive hemifacial atrophy)

First reported by Romberg in 1846.

Characterized by slowly progressive atrophy of the soft tissue of half of the face. It is manifested as progressive wasting of subcutaneous fat, atrophy of the skin, cartilage, bone and muscle.

Etiology

Primary factor is cerebral disturbance leading to increased and unregulated sympathetic nervous system.

Local trauma, infection
Genetic alterations.

Clinical Features

Painless cleft on the midline of the face or forehead.

Atrophy of the skin, subcutaneous fat, muscle, bone, cartilage, alveolar bone. Neurological disturbances may be seen.

Oral Manifestations

Incomplete root formation
Delayed eruption
Difficulty in mastication

Treatment and Prognosis

No specific treatment.

DEVELOPMENTAL DISTURBANCES OF THE LIPS AND PALATE

VAN DER WOUDE'S SYNDROME

(Cleft lip syndrome, lip pit syndrome, dimpled papillae of the lip)

It is an autosomal dominant syndrome consisting of a cleft lip or cleft palate and distinctive pits of the lower lips.

Etiology

Prominent feature of this syndrome is orofacial anomalies. This is due to abnormal fusion of palate and lips at days 30-50 post-conception.

Clinical Features

- Incidence is 1 in 100,000 people.
 - Affects both generation equally.
 - Cleft lip and cleft palate is commonly seen. Unilateral or bilateral involvement is seen.
 - Hypernasal voice and cleft or bifid uvula is the clue for diagnosis.
 - Lower lip pits are quite distinctive and medial on the vermillion portion of the lower lip.
 - Extraoral manifestations
 - Rare but include limb anomalies, popliteal webs, brain anomalies, accessory nipples, congenital heart diseases can be seen.

Treatment and Prognosis

- Surgical repair of the cleft lip and palate in cases of cosmetic reasons.

CONGENITAL LIP AND COMMISSURAL PITS AND FISTULAS

Etiology

- May result from notching of the lip at an early stage of development.
- Fixation of the tissue at the base of the notch.
- Failure of complete union of the embryonic lateral sulci of the lip.
- Occur at the site of horizontal facial cleft and may represent defective development of the embryonic fissure.

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Clinical Features

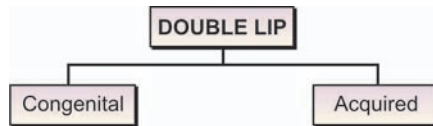
- Unilateral or bilateral depression or pit that occurs on the vermillion surface of lip.
- Sparse mucus secretion may exude from the base of the pit.
- Lip appears swollen, accentuating the appearance of the pits.

Treatment

- Surgical excision has been recommended.

DOUBLE LIP

- It is an anomaly characterized by a fold of excess tissue on the inner mucosal aspect of the lip.



Clinical Features

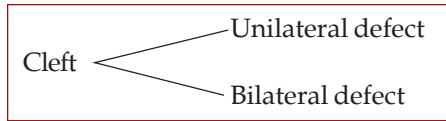
- Both upper and lower lips are involved.
- When the upper lip is sensed, the double lip resembles a cupid's bow.
- Occurrence of acquired double lip in association with blepharochalasis and nontoxic thyroid enlargement is known as "**Ascher's syndrome**".
- Blepharochalasis is drooping of tissue between the eyebrow and the edge of upper eyelid so that it hangs loosely over margin of the lid.

Treatment

- No treatment is necessary except for cosmetic purposes or functions involving speech and mastication.

CLEFT LIP AND CLEFT PALATE

- Classification of cleft lip
 - Unilateral incomplete.
 - Unilateral complete.
 - Bilateral incomplete.
 - Bilateral complete.



- Mandibular cleft lip is an extremely rare condition that occurs in the midline of the lowerlip.
- It is due either failure of the copula to give rise to mandibular arch or to persistence of the central groove of the mandibular process.
- Maxillary cleft lip is thought to be due to failure of the globular portion of the median nasal process to unite properly with the lateral nasal and maxillary process.
- Cleft may also be due not to an actual lack of union of the processes but rather to a failure of mesodermal penetration and the obliteration of the ectodermal grooves separating these mesodermal masses that actually constitute the facial processes.
- Cleft palate appears to represent a disturbance in the normal fusion of the palatal shelves.
- Failure to unite due to lack of forceful interference by the tongue, disparity in the size of the parts involved.

Etiology

- Heredity is undoubtedly one of the most important factors to be considered.

Environmental Factors

- Genetic in origin.
- Physiologic, emotional or traumatic stress may play a significant role.

Other Factors

- A defective vascular supply to the area involved.
- A mechanical disturbance in which the size of the tongue may present the union of parts.
- Circulating substances such as alcohol and certain drugs.
- Infections.
- Lack of inherent developmental force.

Clinical Features

- Unilateral cleft lip involves only one side of the lip or both sides of the lip.
- The latter type has given rise to term harelip.
- Cleft lip and cleft palate are somewhat more common in boys than in girls.
- Cleft occurs about three times more frequently on the left side than on the right.
- Cleft of the hard palate extends anteriorly through the alveolar ridge and lip, producing a complete cleft in the lip, ridge and palate.
- Isolated cleft palate is associated with congenital heart diseases, polydactylism and syndactylism, hydrocephalus, microcephalus, club foot, supernumerary ear, hypospadias, spina bifida, hypertelorism and mental deficiency.
- A median maxillary anterior alveolar cleft might be due to precocious limitation of the growth of the primary ossification centers on either side of the midline at the primary palate or to their subsequent failure to fuse.

Clinical Significance

- Eating and drinking are difficult because of regurgitation of food and liquid through nose.

Treatment

- Surgery can correct cleft palate. Operation to close the cleft is not usually carried out until the patient is 18 months old.

CHEILITIS GLANDULARIS

- Occurs mostly in adult, the lower lip becomes enlarged, firm and finally everted.

Etiology

- Unknown etiology, but it can be due to:
 - Chronic exposure to sun, wind and dust.
 - Use of tobacco.

Clinical Features

- Labial salivary glands become enlarged and sometimes nodular.
- Orifices of the secretory ducts are inflamed and dilated appearing as small red macules on the mucosa three basic types of cheilitis glandularis are seen:
 - Simple type
 - Superficial suppurative type
 - Deep suppurative type.
- Simple type is characterized by multiple painless pinhead sized lesions with central depressions and dilated canals.
- This type may be transformed into superficial suppurative type characterized by painless swelling, induration, crusting, superficial and deep ulcerations of the lip. (Baelz's disease).
- Deep suppurative type (Cheilitis glandularis apostematosa, myxadenitis labialis) is a deep seated infection with abscesses and fistulous tracts that eventually form scars.

Treatment

- Because of relatively high incidence of associated malignancy, a vermillionectomy or surgical stripping of the lip has been recommended.

CHEILITIS GRANULOMATOSA

(Mieschers-Melkersson-Rosenthal syndrome)

Clinical Features

- There is a diffuse swelling of the lips, especially lower lip. Swelling is usually soft and exhibits no pitting upon pressure.
- Scaling, fissuring, erythematous vesicles or pustules present.
- Appears in association with facial paralysis and scrotal tongue called *Melkersson-Rosenthal syndrome*

Histologic Features

- Chronic inflammatory cell infiltrate particularly peri and paravascular aggregations of lymphocytes, plasma cells and histiocytes and focal noncaseating granuloma formation with epithelioid cells and Langhans' type of giant cells.

HEREDITARY INTESTINAL POLYPOSIS SYNDROME

(Peutz-Jeghers syndrome)

- Pigmented spots on the face, oral cavity and sometimes the hands and sometimes the hands and feet.

Clinical Features

- Melanin pigmentation of the lips and oral mucosa is present from birth and appears as small brown macules measuring 1-5 mm in diameter.
- Buccal mucosa is frequently involved.
- On the face the spots tend to be grouped around the eyes, nostrils and lips,
- Lower lip is almost involved.
- Facial pigmentation tends to fade later in life.
- Oromucosal pigmentation includes melanin pigmentation such as:
 - Local and ethnic pigmentation.
 - Oral pigmentary manifestations of systemic diseases.
 - Pigmentary disturbances associated with pharmaceuticals and other chemicals.
 - Benign and malignant pigmented neoplasms.
- Intestinal polyps are distributed through the entire intestine.
- Patients have frequent episodes of abdominal pain and signs of minor obstruction.
- Role of dentist in detecting this syndrome is important through a tentative diagnosis based on the oral and paraoral manifestations.

DEVELOPMENTAL DISTURBANCES OF ORAL MUCOSA

FOCAL EPITHELIAL HYPERPLASIA

(Heck's Disease)

It is the one of the most contagious oral papillary lesions caused by HPV (human papilloma virus).

Clinical Features

- Primarily occurs in children but may appear in young and middle aged adult.

- No gender predilection.
- Involves the labial, buccal and lingual mucosa mainly but gingiva and soft palate can be affected.
- Characterized by broad based or slightly elevated, well demarcated plaques. Frequently papillary in nature, relatively smooth surfaced, flat topped lesions.
- Appears as cobblestone or fissured appearance.

Histologic Features

- Considerable focal acanthosis of the oral epithelium.
- The thickened mucosa extends upward, not down into underlying connective tissues, hence the adjacent normal rete ridges.
- Ridges are widened and club shaped.

Treatment and Prognosis

Conservative excisional biopsy

FORDYCE'S GRANULES

(Fordyce's Disease)

- Also called Fordyce's disease and sebaceous nevi.
- Not a disease of oral mucosa. It is a developmental anomaly characterized by heterotopic collections of sebaceous glands at various sites in the oral cavity.
- This occurs due to inclusion of sebaceous glands in the oral cavity of ectoderm during development of the maxillary and mandibular processes of embryonic life.

Clinical Features

- Appears as small yellow spots, either seen individually or forming a large plaques, often projecting slightly above the surface of the tissue.
- Most frequently seen as a bilaterally symmetrical pattern. On the cheek mucosa, opposite the molar teeth.

Other Sites

- Inner surface of the lips.
- Retromolar region lateral to the anterior faucial pillar.
- Occasionally on the tongue, gingiva, frenum and palate.

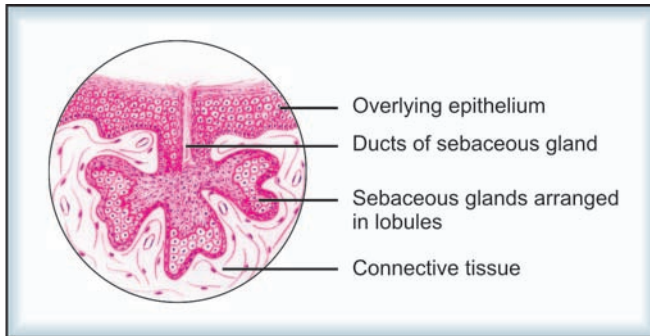


Fig. 1.1: Fordyce's granules

- Current research states that it can be seen in esophagus, the female genitalia including the uterine cervix, the nipples, the palms and soles, the parotid glands, the larynx and the orbit.
- Incidence of oral condition is 80%.
- No significant difference in occurrence between the genders or races.

Histologic Features

- Glands are usually superficial and may consist of only a few or a great lobules, all grouped around one or more ducts which open on the surface of the mucosa.
- Ducts may show keratin plugging.

Treatment

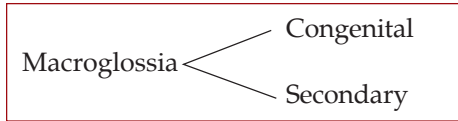
- No treatment required.
- Very rarely a benign sebaceous gland adenoma and keratin filled pseudocyst develops.

DEVELOPMENTAL DISTURBANCES OF THE TONGUE

MICROGLOSSIA

- Is a rare congenital anomaly manifested by the presence of a small or rudimentary tongue.
- When tongue is completely absent at birth, the condition is known as aglossia.

MACROGLOSSIA



- Congenital macroglossia is due to an overdevelopment of the musculature which may or may not be associated with generalized muscular hypertrophy or hemihypertrophy.
- Secondary macroglossia may occur as tumor of the tongue such as diffuse lymphangioma or hemangioma, from neurofibromatosis, blockage of efferent lymphatic vessels, in cases of malignant neoplasms.
- In cases of acromegaly it is due to hyperpituitarism in the adult, cretinism, congenital hypothyroidism; Beckwith's hypoglycemic syndrome.
- Macroglossia may produce displacement of teeth malocclusion.

Treatment

- Removal of the primary cause.
- Surgical trimming.

ANKYLOGLOSSIA

(Tongue tie)



- Fusion between the tongue and floor of the mouth.
- Usually result of a short lingual frenum of one which is attached too near the tip of the tongue.
- There is restricting movement of tongue, exhibit speech difficulties in pronunciation of certain consonants and diphthongia.

Treatment

- Treated surgically by clipping the frenum.

CLEFT TONGUE OR BIFID TONGUE

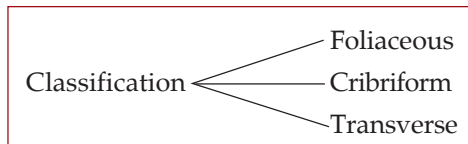
- Is a rare condition that is apparently due to lack of merging of the lateral lingual swellings of this organ.

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- Manifested as a lay groove in the midline of the dorsal surface.
- Results because of incomplete merging and failure of groove obliteration by underlying mesenchymal proliferation.
- It is found in association with orofacial digital syndrome.

FISSURED TONGUE

(*Scrotal tongue, Lingua plicata*)



- Malformation manifested clinically by numerous small furrows or grooves on the dorsal surface, radiating out from a central groove along the midline of the tongue.
- It is not a developmental malformation, the incidence of this condition increases with age.
- Associated with some extrinsic factor such as chronic trauma or vitamin deficiencies.

Treatment

- It is usually painless except in occasional cases in which food debris tends to collect in the grooves and produce irritation.
- Material may be removed by stretching and flattening the fissures and using a tooth brush or gauze sponge to cleanse the surface.

MEDIAN RHOMBOID GLOSSITIS

(*Central papillary atrophy of the tongue*)

- Congenital abnormality which is presumable due to failure of the fusion of tuberculum impair to retract or withdraw before fusion of the lateral halves of the tongue, so that a structure devoid of papillae is interposed between them.
- An etiologic relationship between median rhomboid glossitis and a localized chronic fungal infection specifically *Candida albicans*.
- Presence of fungal hyphae in histology sections of biopsies.
- Common among diabetes.

Clinical Features

- Clinically as an ovoid, diamond or rhomboid shaped reddish patch or plaque on the dorsal surface of tongue anterior to circumvallate papillae.
- A flat or slightly raised area and mamelonated. It has no filiform papillae.
- Occurs three times more frequently in men than women.

Histologic Features

- Loss of papillae with varying degrees of hyperparakeratosis.
- Proliferation of spinous layer with elongation of the rete ridges which may branch and anastomose.
- Lymphocytic infiltration within connective tissue.
- Numerous blood vessels and lymphatics.
- Degeneration and hyaline formation within the underlying muscle.
- Fungal hyphae are found in the parakeratin or in superficial spinous layer of epithelium.
- Best visualized by Periodic acid schiff (PAS) stain.

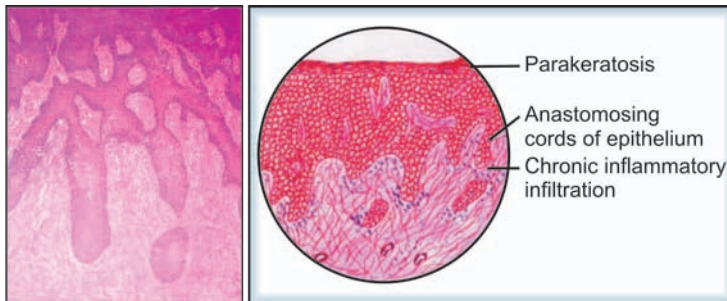


Fig. 1.2: Median rhomboid glossitis

Treatment

- Antifungal agents like nystatin and amphotericin B can be used.

BENIGN MIGRATORY GLOSSITIS

(Geographic tongue, wandering rash, glossitis areata exfoliativa, erythema migrans)

- May be related to emotional stress.

Clinical Features

- Multiple areas of desquamation of the filiform papillae of the tongue in an irregular circinate pattern.
- Central portion of the lesion appears inflamed while the border is outlined by a thin, yellowish white line or band.
- Fungiform papillae persist in the desquamated areas small, elevated red dots.
- Areas of desquamation remains for a short time in one location and heal and appear in another thus giving rise to the idea of migration.
- “Ectopic geographic tongue” or erythema circinate is located at other sites in the oral cavity, like buccal mucosa, gingiva, palate, lips and floor of the mouth.

Histologic Features

- Filiform papillae are lost. Hyperparakeratosis and acanthosis seen.
- Migration of (PMNL) polymorphonuclear leukocytes and lymphocytes into the epithelium producing degeneration of epithelial cells and microabscess formation near the surface.
- Inflammatory cell infiltration of underlying connective tissue chiefly neutrophils, lymphocytes and plasma cells are seen.

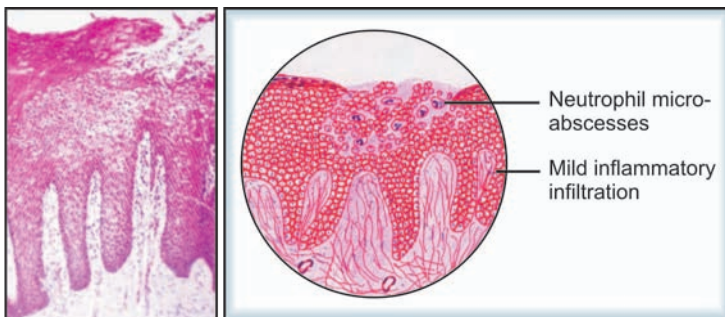


Fig. 1.3: Benign migratory glossitis

Treatment

- Heavy therapeutic doses of vitamins have been used.

HAIRY TONGUE

(Lingua nigra, Black hairy tongue)

Clinical Features

- Condition is characterized by hypertrophy of filiform papillae of the tongue, with lack of normal desquamation which is extensive and form a thick matted layer on dorsal surface.
- Color of papillae may vary from yellowish white to brown or even black depending upon staining by extrinsic factors as tobacco, foods, medicines, chromogenic organisms.

Etiology

- Unknown etiology.
- Certain microorganisms particularly fungi might be exciting factor.
- Many different types of organisms including *Candida albicans* may be cultured from scrapings of the papillae.
- Systemic disturbances like anemia, gastric upsets are responsible for hairy tongue.
- Oral use of certain drugs (sodium perborate, sodium peroxide, antibiotics such as penicillin and aureomycin), heavy smoking.
- Development of hairy tongue is frequently seen in patients who have had extensive X-ray radiation of head and neck.

Treatment

- Benign condition. So, treatment is empirical because of unknown etiology.
- Tongue may be brushed with a toothbrush to promote desquamation and remove the debris.

DEVELOPMENT DISTURBANCES OF SALIVARY GLAND

ATRESIA

It is defined as congenital occlusion or absence of one more of the major salivary gland ducts is an exceedingly rare condition. When it occurs it may result in the formation of a retention cyst or produce a relatively severe xerostomia.

ABERRANCY

Because of widespread distribution of accessory salivary gland it is very difficult to define the condition of aberrancy.

As these salivary glands are common in lips, palate buccal mucosa floor of the mouth, aberrancy can be construed as simply that condition in which these glands are found farther from their own usual location.

No clinical significance to be attached in any event other than they may be the site of development of a retention cyst or neoplasm. Occasional cases have been reported with salivary gland tissue present within the body of the mandible.

It has been found that this glandular tissue anatomically communicated with the normal submaxillary or sublingual gland through a stalk or pedicle of tissue which perforate the lingual cortical plate.

For this reason this aberrancy represents only an extreme example of the condition known as the developmental lingual mandibular salivary gland depression.

APLASIA

(Agenesis)

- Glands or groups of glands may be missing unilaterally or bilaterally.

Classical Features

- Xerostomia or dry mouth.
- Oral mucosa appears dry, smooth, pebbly, cracking of the lips and fissuring of the corners of the mouth.
- Collection and stagnation of food debris around the teeth resulting in rampant dental caries and early loss of deciduous and permanent teeth.

Treatment

- Institution of oral hygiene to prevent dental caries.

XEROSTOMIA

(Dryness of the Mouth)

- Is a clinical manifestation of salivary gland diseases.

Clinical Features

- Patient complains of a dry or burning sensation.
- Severe alterations in mucous membranes and the patient have extreme discomfort.
- Mucosa appears dry atrophic, inflamed, pale and translucent.
- Soreness, burning and pain of the mucous membrane and tongue.

Etiology

- Associated with an emotional reaction; with blockage of duct by calculus.
- With acute or chronic infection of the salivary glands.
- Administration of various drugs such as atropine or various antihistamine drugs, causes transient or partial chronic obstruction.
- Salivary gland aplasia.
- X-ray radiation.
- Administered in the treatment of a tumor, induces prompt xerostomia.
- Vitamin deficiency
- Affects specialized epithelium.
- Results in squamous metaplasia of the ductal epithelium with retention of salivary secretion.
- Sicca syndrome or keratoconjunctivitis sicca is caused by vitamin A deficiency.
- Riboflavin and nicotinic acid deficiencies causes xerostomia.
- Sjögren's syndrome
- Xerostomia occurs because of distinction and atrophy of acinar tissue of the salivary glands.
- Miscellaneous
- Pernicious anemia, iron deficiency anemias.
- Hemorrhage, excessive sweating, diarrhea, vomiting, polyuria, organic lesions of the nervous system.

Clinical Significance

- Rampant dental caries and subsequent loss of teeth.
- Have difficulty with artificial dentures.
- Dental appliances are disagreeable.

Treatment

- Depends upon the nature of the disease. Etiology should be removed. Symptomatic treatment given.

DEVELOPMENTAL LINGUAL MANDIBULAR SALIVARY GLAND DEPRESSION

(Static bone cavity, static bone cyst, latent bone cyst, Stafne cyst or defect)

- First recognized by Stafne in 1942.
- Developmental inclusion of glandular tissue within or adjacent to the lingual surface of the body of mandible in a deep, well circumscribed depression.

Radiological Features

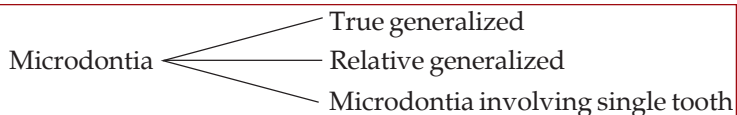
- Appears as an ovoid radiolucency situated between mandibular canal and the inferior border of the mandible. Commonly seen in the second or third molar area just anterior to the angle.
- Occasionally bilateral.
- Depression may be due to actual presence of salivary gland anathion the mandible during embryonic development or may be an indentation on the lingual surface.

DEVELOPMENTAL DISTURBANCES OF TEETH

(Disturbances in Size of Teeth)

MICRODONTIA

- It is used to describe teeth which are smaller than normal.



- In true generalized microdontia, all the teeth are smaller than normal. e.g.: Pituitary dwarfism.
- In relative generalized microdontia, normal or slightly smaller than normal teeth are present in jaws that are somewhat larger than normal.
- Microdontia involving a single tooth, affects mostly the maxillary lateral incisor and the third molar, e.g. Peg lateral.

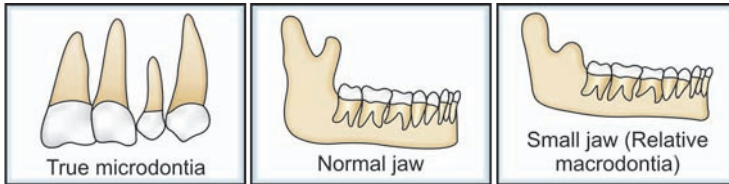
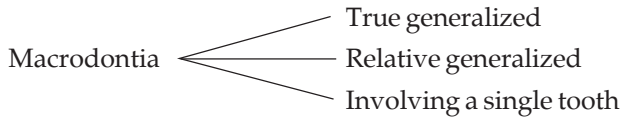


Fig. 1.4: Microdontia

MACRODONTIA

- Refers to teeth larger than normal.



- True generalized: All teeth are larger than normal, e.g.: pituitary gigantism.
- Relative generalized: Presence of slightly larger than normal teeth in small jaws.
- Macrodontia involving single tooth is common.
- In hemihypertrophy of the face it can be seen but its not a true type.

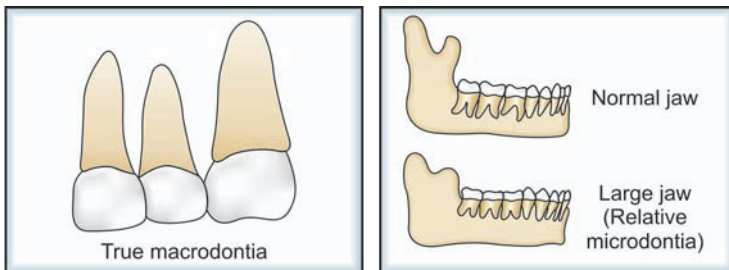


Fig. 1.5: Macrodonia

DEVELOPMENTAL DISTURBANCES IN SHAPE OF TEETH

GEMINATION

- Anomalies which arise from an attempt at division of a single tooth germ by an invagination, with resultant incomplete formation of two teeth.
- The structure is usually one with two completely or incompletely separated crowns that have a single root and root canal.
- Seen in deciduous as well as permanent dentition.
- The term “twinning” is used to designate the production of equal structures by division resulting in one normal and one supernumerary tooth.



Fig. 1.6: Gemination

FUSION

- Arise through union of two normally separated tooth germs.
- Depending on stage of development of teeth at the time of union, fusion may be complete or incomplete.
- Some physical force or pressure produces contact of the developing teeth and their subsequent fusion occurs.
- If contact occurs early before calcification begins, the two teeth may be completely united to form a single large tooth.
- If contact of teeth occur when a portion of the tooth crown has completed its formation there may be union of roots.
- Tooth may have separate or fused root canals and the condition is common in deciduous as well as permanent dentition.



Fig. 1.7: Fusion

- Possible clinical problems related to appearance, spacing and periodontal conditions have been noticed.

CONCRESCENCE

- A form of fusion which occurs after root formation has been completed.

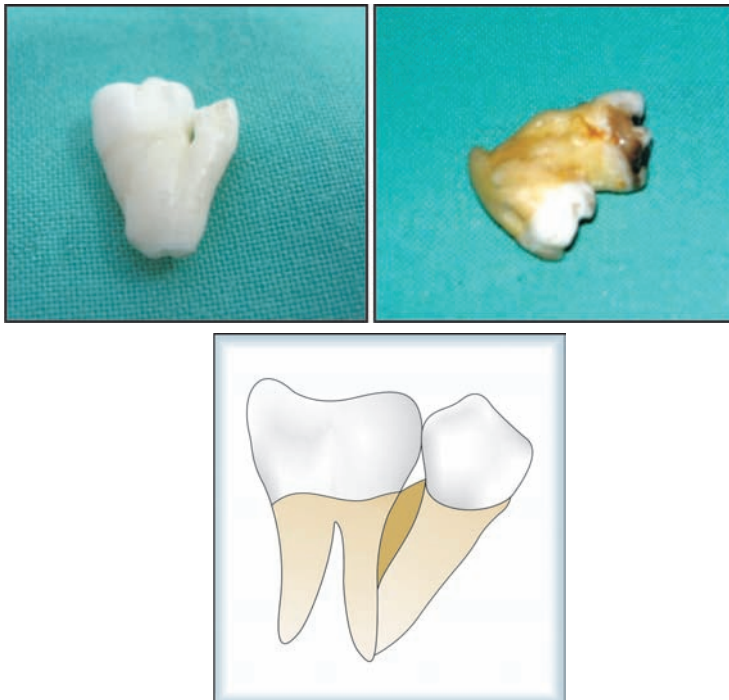


Fig. 1.8: Concrecence

- The teeth are united by cementum only.
- Thought to arise as a result of traumatic injury a crowding of teeth with resorption of interdental bone so that the two roots are in approximate contact and become pushed by deposition of cementum between them.
- May occur before or after teeth have erupted.
- Diagnosis is by roentgenographic examination.
- With fused teeth the extraction of one may result in extraction of the other.

DILACERATION

- Refers to an angulation or a sharp bend or curve in the root or crown of a formed tooth.
- It is thought to be due to trauma during the period in which the tooth is forming with result that the position of calcified portion of the tooth is changed and the remainder of the tooth is formed at an angle.
- Curve or bend may occur anywhere along the length of the tooth, at the cervical portion, at other times midway along the root or even just at the apex of the root, depending upon the amount of root formed when the injury occurred.
- Dilacerated teeth present difficult problems at the time of extraction.

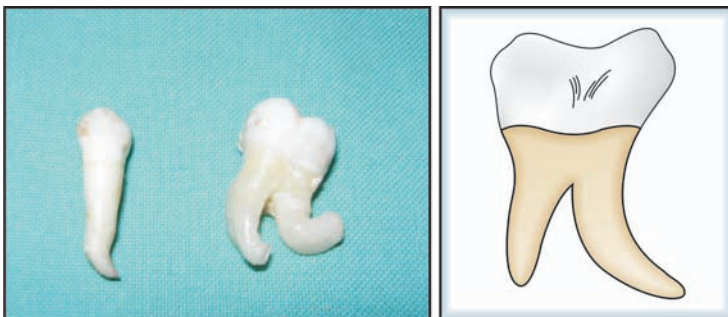


Fig. 1.9: Dilacerations

TALON CUSP

- An anomalous structure resembling an eagles talon projects lingually from the cingulum areas of a maxillary and mandibular permanent incisor.
- The cusp blends smoothly with the tooth except that there is a deep developmental groove where the cusp blends with the sloping lingual tooth surface.
- It poses problems for the patient in terms of esthetics, caries control and occlusal accommodation.
- Prevalent in persons with Rubinstein-Taybi syndrome.

DENS IN DENTE

(*Dens invaginatus, dilated composite odontome*)

- Is a developmental variation which is thought to arise as a result of an invagination in the surface of a tooth crown before calcification has occurred.

Causes

- Increased localized external pressure, focal growth retardation and focal growth stimulation in certain areas of tooth bud.
- Permanent maxillary lateral incisors are mostly involved.
- Maxillary central incisor are sometimes involved.

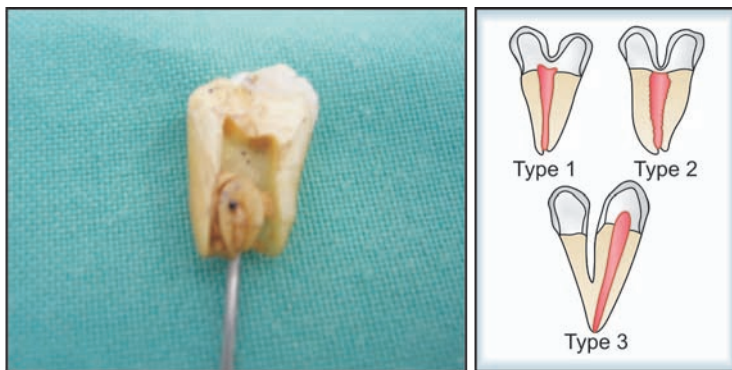


Fig. 1.10: Dens invaginatus (dens indente)

Radiological Features

- It is recognized as a pear shaped invagination of enamel and dentin with a narrow constriction at the opening on the surface of the tooth and closely approximating, the pulp in its depth. Food debris may become packed in this area with resultant caries and infection of the pulp.
- Dens in dente may exhibit an invagination that extends nearly to the apex of the root.

DENS EVAGINATUS

(Occlusal tuberculated premolar, Leong's premolar, evaginated odontome, occlusal enamel pearl)

- Is a developmental condition that appears clinically as an accessor cusp or a globule of enamel on the occlusal surface between the buccal and lingual cusps of premolars, unilaterally or bilaterally.
- Pathogenesis of the lesion is thought to be proliferation and evagination of an area of the inner enamel epithelium and subjacent odontogenic mesenchyme into the dental organ during early tooth development.
- This extra cusp may contribute to incomplete eruption, displacement of teeth or pulp exposure with subsequent infection following occlusal wear off.

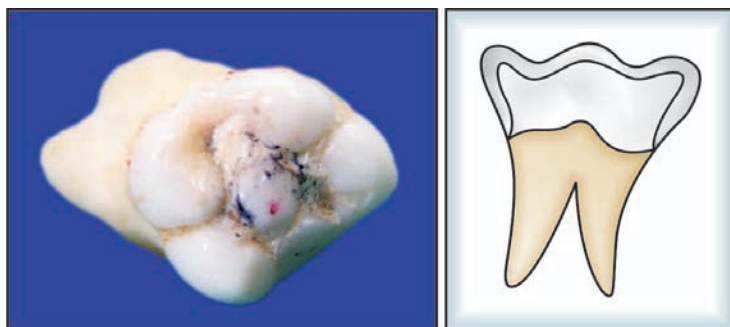


Fig. 1.11: Dens evaginatus

TAURODONTISM

(Bull like teeth)

- Dental anomaly in which the body of the tooth is enlarged at the expense of the roots.
- Shaw classified taurodont into
 - Hypotaurodont
 - Mesotaurodont
 - Hypertaurodont

Causes of Taurodontism

- Failure of epithelial sheath to invaginate at the proper horizontal level.
- Specialized or retrograde character.
- Primitive pattern.
- Mendelian recessive trait.

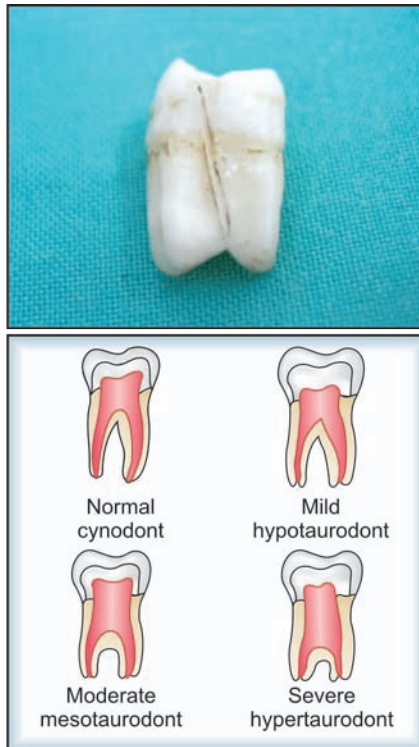


Fig. 1.12: Taurodontism (bull like teeth)

- Atavistic feature.
- Mutation resulting from odontoblastic deficiency during dentinogenesis of the roots.
- It is associated with **Klinefelter's syndrome** (males whose sex chromosome constitution includes one or more extra X chromosomes).

Clinical Features

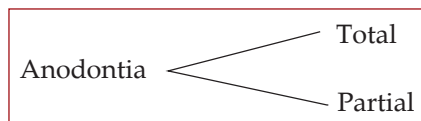
- May affect either deciduous or permanent dentition.
- Teeth involved are molars.
- It may be unilateral/bilateral.

Radiological Features

- Involved teeth are rectangular in shape.
- Pulp chamber is extremely large with greater apico-occlusal height.
- Pulp lacks the usual constriction at the cervical of the tooth and roots are short.
- Bifurcation or trifurcation is only a few mm above the apices of roots.

DEVELOPMENTAL DISTURBANCES IN NUMBER OF TEETH

ANODONTIA



- Total anodontia in which all teeth are missing may involve both deciduous and permanent dentition. It is associated with hereditary ectodermal dysplasia.
- Induced or false anodontia occurs as result of extraction of all teeth.
- Pseudoanodontia is applied to multiple unerupted teeth.
- True partial anodontia (hypodontia or oligodontia).
- All four first molars are missing.
- Maxillary lateral incisors, maxillary and mandibular second premolars are missing.

- Congenitally missing deciduous teeth usually involve the maxillary lateral incisor.
- The etiology of missing teeth is that it is actually the result of one or more point mutations in a closely linked polygenic system transmitted as an autosomal dominant pattern with incomplete penetrance and variable expressivity.

SUPERNUMERARY TEETH

- Develop from a fluid tooth bud arising from the dental lamina near permanent tooth bud or from splitting of the permanent but itself.
- Most common tooth is mesiodens.
- Maxillary 4th molar is the second most supernumerary tooth.
- Other supernumerary teeth are maxillary paramolars, mandibular premolars and maxillary lateral incisors.
- It may be erupted or impacted.
- Frequently cause malposition of adjacent teeth or present their eruption.
- Found in cleidocranial dysplasia, Gardner syndrome.

DEVELOPMENTAL DISTURBANCES OF STRUCTURE OF TEETH

ENVIRONMENTAL ENAMEL HYPOPLASIA

Caused by environmental factors.

Either primary or permanent dentition is involved.

Both enamel and dentin are affected.

Causes

Nutritional deficiencies

Exanthematous diseases

Congenital syphilis

Hypocalcemia

Birth injury, prematurity, Rh incompatibility

Local infection/trauma

Ingestion of chemicals

Idiopathic

Types

- Mild – few small grooves, pits or fissures in enamel surface.
- Moderate – rows of deep pits arranged horizontally across the tooth.
- Severe – considerable portion of enamel may be lost.

Clinical Features

Due to nutritional deficiency

Mainly deficiency of vitamin A, C and D during tooth formation.

Usually pitting type of hypoplasia is seen.

Involve those teeth that form within the 1st year after birth.

Due to exanthematous fevers

Measles, chickenpox, scarlet fever are the common causes.

Ameloblasts being one of the most sensitive group of cells in terms of metabolic function are easily affected by any systemic diseases.

Due to congenital syphilis

Pathognomonic appearance is seen.

Involves maxillary and mandibular permanent incisors and 1st molars.

Incisors show “Hutchinson’s teeth” characterized by screw driver shaped, mesial and distal surfaces of crown taper towards the notched incisal edge.

Molars show “Mulberry molars” characterized by irregular crowns and enamel of the occlusal 1/3rd arranged in an agglomerate mass.

Due to hypocalcemia

When serum calcium level falls below 6 mg/100 ml, pitting variety of enamel hypoplasia is seen.

Due to birth injuries

Hypoplasia is common in prematurely born children.

Children suffering from Rh hemolytic diseases at birth showed recognized staining of teeth.

Generally seen in the enamel formed after deciduous dentition

Due to local infections

Only single tooth is involved.

Turner's teeth – any degree of hypoplasia (mild, moderate and severe).

Infection of periapical region of deciduous teeth affect the ameloblastic layer of secondary dentition resulting in hypoplastic crowns.

Due to chemicals

Mainly due to fluorides

Mottled enamel is seen characterized by ingestion of fluoride in drinking water during the tooth formation.

Disturbance of the ameloblasts during formative stage results in defective or deficient enamel matrix.

Clinical features varies from occasional white flecking or spotting to white opaque areas involving more of tooth surface, pitting and brownish staining of surface and corroded appearance of the teeth.

Due to idiopathic factors

Ameloblast being a sensitive cell gets affected easily by even illness or some mild systemic disturbances, which is not significantly remembered by the patients.

AMELOGENESIS IMPERFECTA

(Hereditary enamel dysplasia, hereditary brown enamel, hereditary brown opalescent teeth)

- It is an ectodermal disturbance since the mesodermal components of the teeth are normal.
- Three basic types of amelogenesis imperfecta are recognized:
 - Hypoplastic type in which there is defective formation of matrix.
 - Hypocalcification (hypomineralization) in which there is defective mineralization of the formed matrix.
 - Hypomaturation type in which enamel crystallites remain immature.

Clinical Features

1. Hypoplastic type

- Enamel has not formed to full normal thickness on newly erupted teeth.

2. *Hypocalcified type*

- Enamel is so soft that it can be removed by prophylaxis instrument.

3. *Hypomaturation type*

- Enamel can be pierced by an explorer point under firm pressure or can be lost by chipping away from the underlying normal appearing dentin.
- Crowns of the teeth may or may not show discoloration. If present it varies between yellow to dark brown.
- It may have a chalky texture or a cheesy consistency. It may be chipped or show depressions.

Histologic Features

- There is a disturbance in the differentiation or viability of ameloblasts in the hypoplastic type and this is reflected in defects in matrix formation up to and including total absence of matrix.
- In hypocalcification type there are defects of matrix structure and mineral deposition.
- In hypomaturation type there are alterations in enamel rod and rod sheath structure.

Treatment

- Improvement of cosmetic appearance.

DENTINOGENESIS IMPERFECTA

(Hereditary opalescent dentin)

- Here the mesodermal portion of the odontogenic apparatus is disturbed.

Classification

- *Type I:* Occurs in families with osteogenesis imperfecta.
It is an autosomal dominant trait with variable expressivity.
- *Type II:* Never occurs in association with osteogenesis imperfecta.
Referred to as hereditary opalescent dentin.
Inherited as an autosomal dominant trait.

- *Type III:* It is of Brandywine type. This is a racial isolate in Maryland.
There is multiple pulp exposures in deciduous teeth.
It is an autosomal dominant.

Clinical Features

- Color of teeth may range from a gray to brownish-violet or yellowish-brown.
- Exhibits a characteristic translucent or opalescent hue.
- There is an abnormal dentinoenamel junction and the scalloping is absent.
- Dentin undergoes rapid attrition and occlusal surfaces are severally flattened.

Radiological Features

- There is partial or total precocious obliteration of the pulp chambers and root canals by continued formation of dentin.
- Enamel is normal while dentin is thin and pulp chambers are enormous. This is shell teeth.
- Most of the teeth exhibit short roots.

Histologic Features

- There is purely a mesodermal disturbance.
- Dentin is composed of irregular tubules often with large areas of uncalcified matrix.
- Tubules tend to be larger in diameter.
- Pulp chamber is obliterated by the continued deposition of dentin.
- Odontoblasts degenerate readily becoming entrapped in the matrix.

Chemical and Physical Features

- Water content is greatly increased (60% above normal). Inorganic content is less than that of normal dentin.

Treatment

- Cast metal crowns on posterior teeth and jacket crowns on anterior teeth have been used.

DENTIN DYSPLASIA

(Rootless teeth)

- Rare disturbance of dentin formation characterized by normal pulpal morphology.
Type I – Radicular dentin dysplasia
Type II – Coronal dentin dysplasia.

Etiology

- Hereditary disease transmitted as an autosomal dominant characteristic.

Clinical Features

- *Type I (Radicular)*: Both dentitions are affected. Teeth exhibit extreme mobility and are commonly exfoliated prematurely or after minor trauma.
- *Type II (Coronal)*: Both dentitions are affected. Deciduous teeth have brown or bluish grey opalescent appearance.

Radiological Features

- *Type I (Radicular)*: In both dentitions roots are short, blunt, conical and malformed, root canals are obliterated. Crescent shaped pulpal remnants may be seen in pulp chamber.
- *Type II (Coronal)*: Exhibit an abnormally large pulp chamber in the coronal portion of the tooth offer described as thistle tube in shape.

Histologic Features

- *Type I (Radicular)*: Apical to the coronal dentin is tubular dentin, most of which obliterates the pulp is calcified tubular dentin, osteodentin and fused denticles. New dentin forms around obstacles and shows an characteristic appearance as lava flowing around boulders.
- *Type II (Coronal)*: Deciduous teeth exhibit amorphous and atubular dentin in the radicular portion. Coronal dentin is normal. Pulp has multiple pulp stones or denticles.

REGIONAL ODONTODYSPLASIA

(Odontogenesis imperfecta, ghost teeth, odontodysplasia, odontogenic dysplasia)

Etiology

- Local vascular defects are involved in the pathogenesis of the condition.

Clinical Features

- Delay or total failure in eruption.
- Shape is markedly altered, irregular in appearance with evidence of defective mineralization.

Radiological Features

- Marked reduction in radiodensity so that the teeth assume “ghost appearance”
- Enamel and dentin are thin and pulp chamber is large.

Histologic Features

- Marked reduction in amount of dentin, widening of predentin layer, presence of large areas of interglobular dentin and an irregular tubular pattern of dentin.

Treatment

- Extraction with restoration by prosthetic appliance.

DISTURBANCES OF GROWTH OF TEETH

- Deciduous teeth erupted into oral cavity seen in infants at birth. These are called natal teeth.
- Neonatal teeth are those teeth erupting prematurely in the first 30 days of life.
- Only 1 or 2 teeth erupt early most often deciduous and mandibular central incisors.
- In cases of endocrine dysfunction (hyperthyroidism) cases it involves the entire dentition.

PREMATURE ERUPTION

- Deciduous teeth that have erupted into the oral cavity are occasionally seen in the infants at birth.
- These are called natal teeth in contrast with neonatal teeth which have been defined as those teeth erupting prematurely in the first 30 days of life.
- Most often the deciduous and mandibular central incisors are involved.
- Etiology of this phenomenon is unknown, although in some instances it follows a familial pattern. In case of the adrenogenital syndrome developing early in life, premature eruption of teeth is sometime seen.
- It has been pointed that premature erupted teeth are often well formed and normal in all respect except that they may be somewhat mobile. The premature eruption of permanent teeth is usually sequelae of the premature loss of deciduous teeth this is best demonstrated in the situation of single deciduous tooth has been loss, with subsequent of permanent tooth.

EMBEDDED AND IMPACTED TEETH

- Individual teeth which are unerupted usually because of lack of eruption force is called embedded teeth.
- Impacted teeth are those prevented from erupting by some physical barrier in the eruption teeth.
- Lack of space due to crowding of dental arches or the premature loss of deciduous teeth with subsequent partial closure of the area.
- Maxillary and mandibular third molars and maxillary cuspids are frequently impacted.

Winter's Classification of Mandibular third Molars

- *Mesioangular impaction:* Third molar lies obliquely in bone, crown pointing in a mesial direction, usually in contact with distal surface of the root or crown of the second molar.
- *Distoangular impaction:* Third molar lies obliquely in bone, crown of the tooth pointing distally towards the ramus.

- *Vertical impaction:* There is lack of space for eruption. It is prevented from erupting by impingement on the distal surface of IInd molar or the anterior border of ramus.
- *Horizontal impaction:* It is in horizontal position with respect to body of mandible.
- Crown may or may not be in contact with the distal surface of second molar.
- Completely impacted tooth is one which lies completely within the bone.
- Partially impacted tooth is not completely encased in bone but lies partially in soft tissue.
- This tooth creates an ideal situation for infection and even dental caries.

ANKYLOSED DECIDUOUS TEETH

(Submerged teeth)

- Are deciduous teeth, most commonly mandibular second molars that have a variable degree of root resorption and then have become ankylosed to the bone.
- This process presents exfoliation and subsequent replacement by permanent teeth.
- After the adjacent permanent teeth have erupted, the ankylosed tooth appears to have submerged below level of occlusion.
- Teeth affected lack mobility even though root resorption is far advanced.
- Upon percussion ankylosed tooth imparts characteristic solid sound in comparison to dull cushioned sound of normal tooth.
- Trauma, infection, disturbed local metabolism, or a genetic influence have been considered.

DEVELOPMENTAL CYST OF ORAL REGION

Nasolabial Cyst

Usually described as a rare fissural cyst that may involve bone secondarily.

Clinical Features

This may cause swelling in the mucolabial fold as well as in the floor of the nose, being located near the attachment of the ala over the maxilla.

Bilateral cases are rare.

This cyst probably originates from the lower anterior part of the nasolacrimal duct rather than from epithelium entrapped in the naso-optic furrow.

Histologic Features

May be lined by pseudostratified columnar epithelium which is sometimes ciliated, often with goblet cells or by stratified squamous epithelium.

Treatment

The cyst should be surgically excised although care must be taken to prevent perforation and collapse of the lesion.

Median Anterior Maxillary Cyst

(Nasopalatine duct cyst, incisive canal cyst)

- Most common type of maxillary developmental cyst.
- Arises from proliferation of epithelial remnants of the nasopalatine duct, an embryologic structure consisting of a duct or cord of epithelial cells lying within in the incisive canal.

Clinical Features

- Can occur at any age, even in the fetus.
- Discovered in the 4- 6th decade.
- They are asymptomatic but become infected and produce pain and swelling and open by a tiny fistula on or near the palatine papilla.

Radiological Features

- Round, ovoid or heart shaped radiolucent area usually bilaterally symmetrical and well outlined.

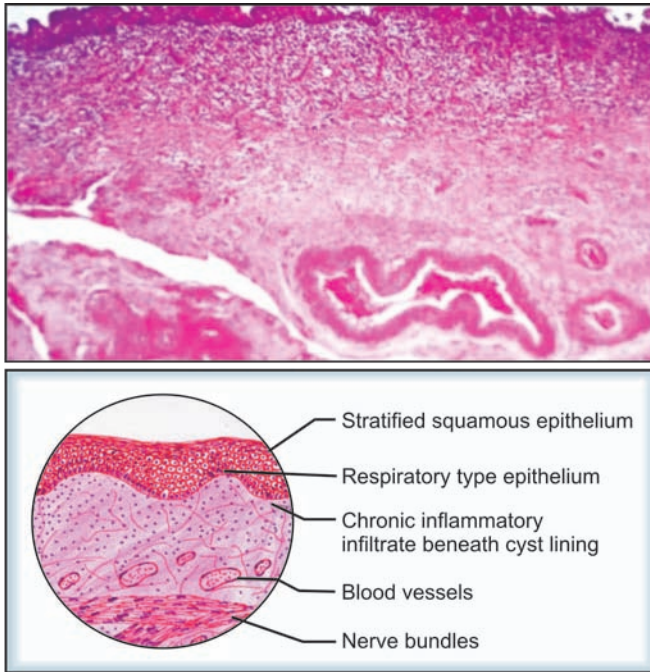


Fig. 1.13: Nasopalatine cyst

- Area appears to lie in the midline between the roots of maxillary central incisors and may cause separation or divergence of roots.

Histologic Features

- Lined by stratified squamous epithelium pseudostratified ciliated columnar epithelium, cuboidal epithelium or any combination.
- Connective tissue wall shows inflammatory cell infiltration.

Treatment

- Surgical excision not necessary.

Globulomaxillary Cyst

- Is found within the bone at the junction of globular portion of medial nasal process and maxillary process. Globulomaxillary fissure is usually seen in between maxillary lateral incisor and cuspid.

- The cyst actually forms in the bone suture between premaxilla and maxilla.

Clinical Features

- Cyst does become infected and the patient may complain of local discomfort or pain in the area.

Radiological Features

- Appears as an inverted pear shaped radiolucent area between the roots of lateral incisor and cuspid causing divergence of roots of these teeth.

Histologic Features

- Is lined by pseudostratified ciliated columnar epithelium remainder of wall is made up of fibrous connective tissue showing inflammatory cell infiltration.

Treatment

- Should be surgically removed, preserving adjacent teeth.

Palatal Cysts of the Neonate

(Palatal and alveolar cysts of newborn)

(Epstein pearls, Bohn's nodules, gingival cyst of the newborn)

- Derived from epithelial remnants of developing palatal salivary glands.
- Found along the median raphe of the hard palate and appeared to be derived from entrapped epithelial remnants along the line of fusion.
- It may be found on alveolar ridges and is derived from remnants of dental lamina and is thus odontogenic in origin. This type is known as dental lamina cyst of newborn.

Clinical Features

- Appear as small, white, raised nodules, usually multiple measuring a fraction of a millimeter to 2 to 3 mm in diameter.
- They tend to cluster along junction of hard and soft palate.

Histologic Features

- Lined by a thin, compressed layer of stratified squamous epithelium with the lumen generally packed with orthokeratin or parakeratin.

Treatment

- No treatment is necessary.

Thyroglossal Tract Cyst

- It is a developmental cyst which may form along the embryonic thyroglossal tract between foramen caecum of tongue and thyroid glands.
- Arises from remnants of this tract that do not become obliterated.

Clinical Features

- Usually occurs in young persons.
- Appears clinically as a firm, cystic, midline mass, varying in size from a few mm to several cm.
- Swelling develops slowly and is asymptomatic.
- If it is present high in the tract it causes dysphagia.
- Cyst may be present in the floor of the mouth near foramen caecum, in the floor of the mouth or inferiorly near thyroid or cricoid cartilage.

Histologic Features

- Thyroglossal tract cyst may be lined by stratified squamous epithelium ciliated type.
- The increased intracystic pressure causes cells become flattened.
- Connective tissue wall of the cyst contain small patches of lymphoid tissue, thyroid tissue and mucous glands.

Treatment

- Complete surgical excision.

EPIDERMOID AND DERMOID CYST

- It is a form of cyst, derived principally from embryonic germinal epithelium.
- These cysts are noted at the floor of the mouth and submaxillary and sublingual areas were common sites.
- Presumed to be derived from enclavement of epithelial debris in the midline during closure of the mandibular and hyoid branchial arches.

Clinical Features

- Arise in the floor of the mouth occur in young adulthood.
- Produces a bulge in the floor of the mouth often causing elevation

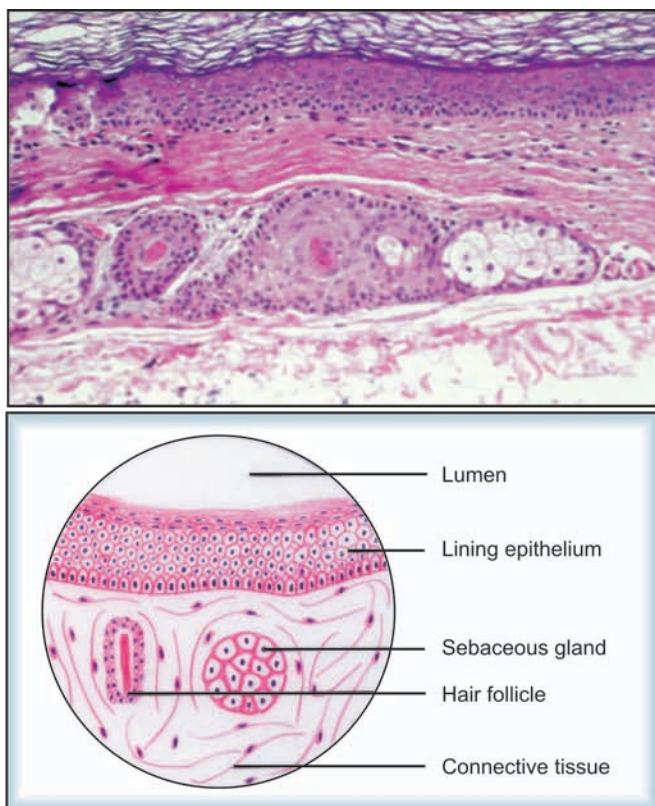


Fig. 1.14: Dermoid cyst

of the tongue and ensuing difficulty in eating and talking if the cyst occurs above geniohyoid muscle.

- If the cyst is situated deeper between geniohyoid and mylohyoid muscles, bulging in submental area is common.
- The cyst has “doughlike” feel to palpation but may be more fluctuant.
- The cyst sometimes becomes infected and develops sinus tracts opening intraorally.
- They may undergo malignant transformation.

Histologic Features

- These cysts are composed only of connective tissue wall lined on the inner surface by a thin layer of stratified squamous epithelium usually showing keratinization.
- Lumen may actually be filled with keratin.
- There may be numerous sebaceous glands and even hair follicles in addition to sweat glands.
- Lumen contains sebaceous material as well as keratin.

Treatment

- Should be removed surgically.

Benign Cervical Lymphoepithelial Cyst

(branchial cleft cyst, lateral cervical cyst, benign cystic lymph node)

- Is a cyst which occurs on the lateral aspect of the neck and is originating from remnants of the branchial arches or pharyngeal pouches.
- This cyst originates through cystic transformation of epithelium entrapped in cervical lymph nodes.

Clinical Features

- Occur in young adult.
- Present as asymptomatic circumscribed movable mass on lateral aspect of the upper neck close to the anterior border of sternocleidomastoid muscle.

- Many cysts occur in neck, angle of mandible, submandibular area and even in preauricular and parotid areas.

Histologic Features

- Lined by stratified squamous epithelium may contain some pseudostratified columnar epithelium.
- Wall of the cyst exhibits lymphoid tissue with a typical lymphnode pattern.
- Cyst may contain thin watery fluid or a thick gelatinous, mucoid material.

Treatment

- Treated by surgical removal.
- Recurrence results, if residual remnants are left or if the lesion is simply aspirated.
- It might develop into a carcinoma.

Dental Caries

Definition

Dental caries is an irreversible microbiologic disease of calcified tissues of teeth characterized by demineralization of the inorganic portion and dissolution of organic substances of tooth which often leads to cavitation.

Epidemiology of dental caries

In Prehistoric Man

- Data about decay in ancient population is with fact that teeth are imperishable on burial sites.
- So preneolithic (12000 BC) periods dolichocephalic skulls did not exhibit caries but skulls from brachycephalic man of the neolithic period contained carious tooth.

In Modern Society

- By about 17th century dental caries became prevalent due to highly industrialized society.
- Main cause of increased incidence is due to dietary factors (civilized foods).

Factors Affecting Caries Prevalence

Race

- Some studies show that blacks have considerably less caries than whites. Racial difference in caries prevalence is due to diet or fluoride exposure.

Age

- Children from families in low socioeconomic groups consistently have greater caries prevalence than their same age group from families at a higher socioeconomic level.

Gender

- Females are commonly affected with caries in permanent dentition due to early eruption of tooth. In deciduous dentition males caries experience is greater.

Familial

- Children of parents with low caries incidence tend to have low caries. Monozygotic twin pair show high concordance of carious site than dizygotic twins.

Etiology of Dental Caries

- The word caries is derived from the Latin word meaning 'rot' or 'decay'.
- Etiology for dental caries is a complicated by many indirect factors. Many theories were put forth but commonly used are acidogenic theory (Miller's chemico-parasitic theory), the proteolytic theory and the proteolysis chelation theory.

Theories of Dental Caries

(A) The Worm Theory

- Caries is caused by worms was evident from writings of Homer who said worms cause toothache.

(B) Endogenous Theory

- According to Greek's vital theory of tooth decay, it is postulated that tooth decay originated like bone gangrene within the tooth itself.

(C) Chemical Theory

- Parmly (1820) observed that dental decay affected externally, not internally, as had been claimed. It was proposed that a chemical agent was responsible for caries. Robertson (1835) proposed that dental decay was by acid, formed by fermentation of food particles around the teeth.

(D) Parasitic Theory

- The parasitic concept was determined by Leber and Rottenstein who proposed that dental caries commenced as a purely chemical process but that living microorganisms continued the disintegration in both enamel and dentin. In addition bacteria were essential to caries, although they suggested an exogenous source of the acid.

(E) Miller's Chemicoparasitic Theory or the Acidogenic Theory

- This is a combination of chemical and parasitic theories, because it states that caries is caused by acid producing microorganism of the mouth.
- It is otherwise called chemoparasitic theory proposed by Miller in 1882.
- In this hypothesis he stated that "Dental decay is a chemo-parasitic dissolution of softened organic residue. In enamel and dentin decalcification it is destroyed totally since inorganic is higher in enamel constituting 96 percent. Three factors play essential role in causing dental caries.

Bacteria + Sugar + Teeth :- produces organic acid which causes caries.

(i) Role of carbohydrates

- Cariogenicity of dietary carbohydrates varies with frequency of ingestion, physical form, chemical composition. Sticky foods,

soft foods which are less fibrous and refined carbohydrates produce caries.

(ii) Role of microorganisms

- Microorganisms play a major role in initiation of dental caries. microorganisms within tubules of decayed teeth are mainly cocci. Microbial flora are *Lactobacilli*, *Streptococcus mutans*, *Streptococcus salivarius*, *Streptococcus mitior*, *Streptococcus sanguis* and *Actinomyces*.

(iii) Role of acids

- Enzymatic breakdown of carbohydrates and acids formed are lactic acid, butyric acid the acid formed causes decalcification and dissolution.

(iv) Role of dental plaque

- Plaque consist of mucin, desquamated cells, microorganisms. Pellicle formation that serves as nutrient for microorganism. Presence of plaque does not necessarily mean carious lesion will develop at same area.

(F) Proteolytic Theory

- Bacteria producing acid from carbohydrate substrate may degrade protein in absence of carbohydrate.
- In one type, organisms invade lamella attack enamel and involve dentin before clinically evident, but in the other type, there is alteration in enamel prior to microorganism invasion.
- Initial decalcification occurs followed by the lysis of organic collagen protein which is called proteolysis.
- Thus proteolysis does not initiate but it plays part in progression of more advanced carious lesion.

(G) Proteolytic Chelation Theory

- This theory proposed by Schatz implies simultaneous microbial degradation of the organic components and dissolution of minerals of tooth by process of chelation.
- Chelation is process of formation of complex substances from metallic ions through coordinate covalent bond which forms highly stable weakly ionized compound.

- In chelation keratinous protein are less stable and formation of complexes of calcium ions.

Factors Causing Caries

- Morphology and position of tooth.
- Constituents, quantity and pH of saliva.
- Dietary factors.
- Systemic factors like pregnancy, heredity and lactation.

CLINICAL CLASSIFICATION OF DENTAL CARIES

(i) Based on Morphology or Anatomical Site of the Lesion on an Individual Tooth

It is classified as:

- Pit and fissure caries
- Smooth surface caries
- Linear enamel caries
- Cervical caries
- Root surface caries.

(ii) Based on Severity and Rate of Progression

- Acute caries
- Chronic caries.

Smooth Surface Caries

- This type develops on the proximal surfaces of teeth or on gingival one-third of facial and lingual surfaces.
- Cervical caries appears on cervical area from occlusal portion of gingival crest to convexity of tooth.

Pit and Fissure Caries

- Develops on occlusal surface of molars and premolars. From the pit, progression of carries starts laterally at DEJ and dentinal involvement without fracture of overhanging enamel. Pulp involvement is delayed.
- Pit and fissure caries appear black or brown and is soft which will catch a fine explorer point.

Secondary Caries

- Discrepancy of restorative material forming microleakage and recurrence of caries is called secondary caries.

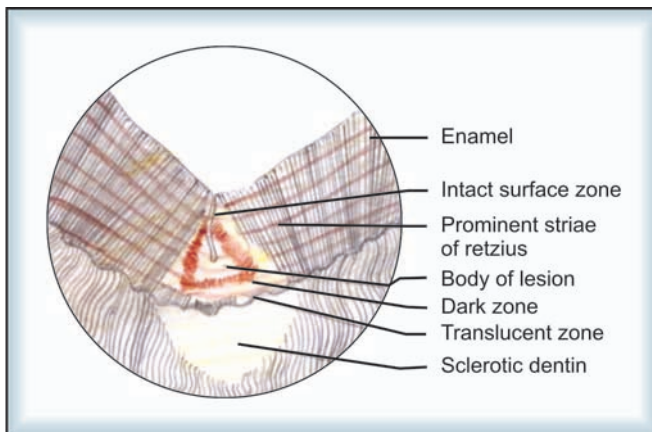
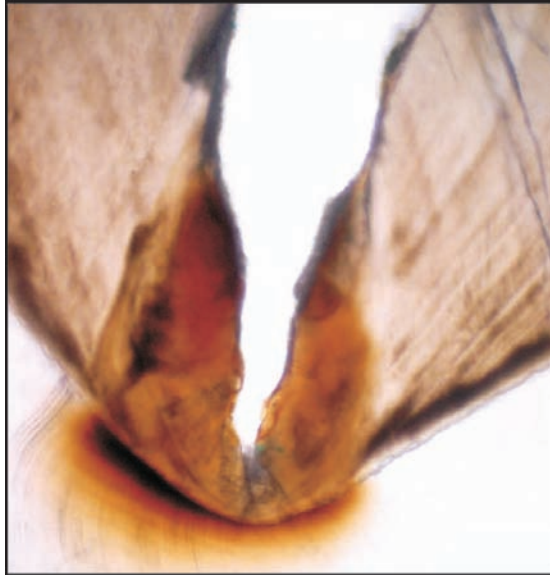


Fig. 2.1: Enamel caries (pit and fissure)

Root Caries

- A soft progressive lesion on the root surface that has lost connective tissue attachment and is exposed to oral environment.
- Root surface caries common in elder patients with gingival recession and exposed root surface.
- Caries of cementum is root caries.

Acute Caries

- Acute caries is a form of caries which is rapid and causes early pulp involvement.
- Pain is prominent feature of acute dental caries.
- Common in children called Nursing Bottle Syndrome.

Chronic Caries

- This type progresses slowly and involves pulp at a slower rate.
- Common in adults.
- Pain is not a common feature because of protection afforded to pulp by secondary dentin.

(A) CARIES OF ENAMEL

Smooth Caries

- It forms cone shaped lesion with apex toward DEJ and base toward surface.

Pit and Fissure Caries

- Cone shaped lesion with apex toward the surface and base towards DEJ.
- First change is loss of prismatic substance, appearance of transverse striations of enamel rods, followed by accentuation of striae of RETZIUS.

HISTOPATHOLOGY OF DENTAL CARIES (CARIES OF ENAMEL)

Histologic Features

Translucent Zone

- Present in advancing front of enamel with high fluoride content no loss of protein.

Dark zone

- Superficial to translucent zone. Positive zone of demineralization.

Body of lesion

- Between dark zone and surface zone layer is relatively translucent, with increase in unbound water and organic content.

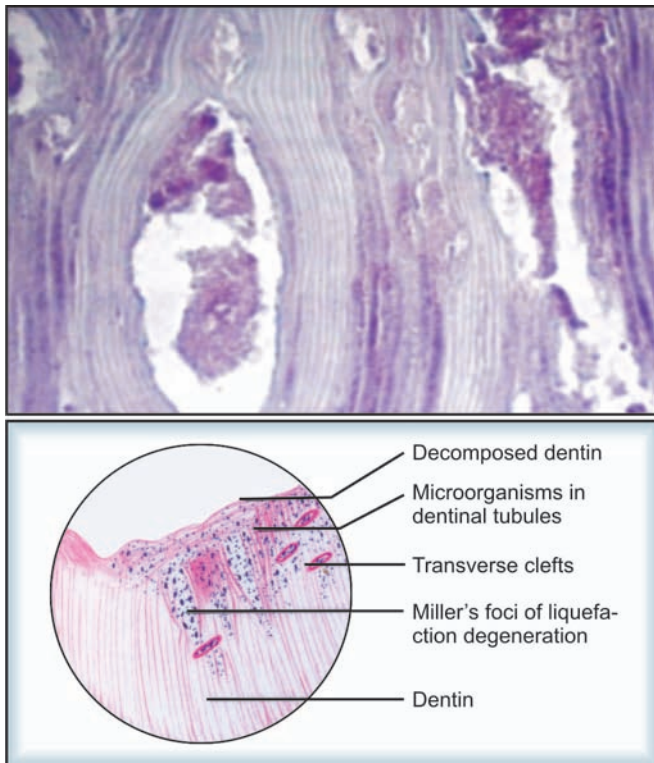


Fig. 2.2: Dentinal caries

Surface zone

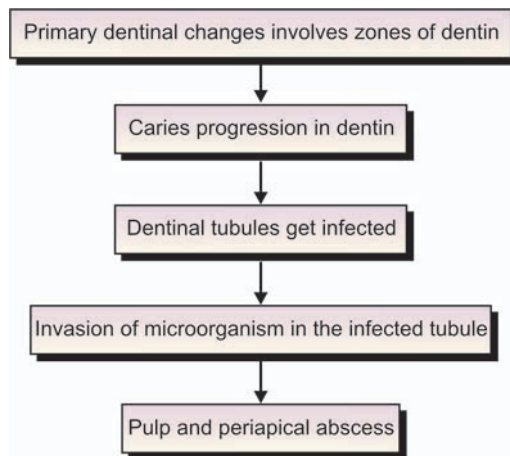
- Only partial demineralization occurs due to resistance by fluoride concentration and insoluble protein in surface layer.

CARIES OF DENTIN**Primary Dentinal Changes**

- ZONE 1 - Zone of fatty degeneration of Tomes fibers.
 ZONE 2 - Zone of dentinal sclerosis due to deposition of calcium salts in tubules.
 ZONE 3 - Zone of decalcification.
 ZONE 4 - Zone of bacterial invasion of intact dentin.
 ZONE 5 - Zone of decomposed dentin.

Secondary Dentinal Changes

- It is slower in rate due to irregularity in tubule arrangement
- Involvement of pulp results in inflammation and necrosis

**METHODS OF CARIES CONTROLS****Chemical Measures**

- Fluoride in water supply, fluoride dentifrices, mouthwash, rinses, topical application of fluoride.
- Chlorhexidine

- Vitamin K supplement
- Antibiotics like penicillin, erythromycin, vancomycin and spiramycin
- Caries vaccine
- Phosphated diet.

Mechanical Measures

- Dental prophylaxis
- Tooth brushing
- Mouth rinsing
- Dental floss
- Irrigators
- Chewing gum
- Pit and fissure sealants.

Diseases of the Pulp and Periapical Tissues

- Dental pulp is a delicate connective tissue consisting of numerous blood vessels, lymphatics, myelinated and unmyelinated nerves and undifferentiated connective tissue cells. Inflammatory condition can also be appreciated in pulpal tissue which is also called pulpitis, which is the most common cause of odontalgia or toothache. Certain anatomic features of this specialized connective tissue act to alter the nature and the course of this response. Due to inflammation, the pressure is increased inside the rigid calcified walls showing hyperemic and edematous phases of inflammation.
- The diseases of the dental pulp occurring directly as sequelae of dental caries.
- The dental pulp is a delicate connective tissue interspersed with tiny blood vessels, lymphatics, myelinated and unmyelinated nerves and undifferentiated connective tissue cells. It reacts to infection or other stimuli by an inflammatory response similar to other connective tissues, but due to certain anatomic features, the nature and course of this response may be altered, e.g. enclosure of pulp within the calcified walls of dentin, blood supply through the tiny apical foramina.

Etiologic Factors of Pulp Disease

- Dental caries in which bacterial invasion of the dentin and the pulp tissue occurs, i.e. Bacterial.
- Tooth fracture which expose the pulp to oral fluids and micro-organisms or bacteremia, i.e. Mechanical.
- Chemical or mechanical injury to the pulp which bring the bacteria circulating in the blood stream to settle out at the sites of pulpal inflammation also called as “Anachoretic pulpitis” chemical.
- Severe thermal change mainly in teeth with large metallic restoration without proper pulpal insulation, i.e. Thermal.
- Electrosurgical current.

Classification of Pulp Disease

- Acute pulpitis.
- Chronic pulpitis.
- Partial or focal pulpitis.
- Total or generalized pulpitis.
- Open pulpitis (pulpitis aperta)- the pulp communicates with the oral cavity.
- Closed pulpitis (pulpitis clausa) no communication of pulp with oral cavity.
- Reversible pulpitis.
- Irreversible pulpitis.

PULPITIS

- The dental pulp lies in a very confined area within the dentin. The active dilation of the arterioles which occurs as an initial response to injury, leads to increased pulpal pressure and secondary compression of the venous return, which can lead to strangulation of the bacterial inflow. The increased pressure appears to be confined to the area of the pulp receiving the noxious stimulus. The increased pulpal pressure combined with the accumulation of mediators can lead to vessel damage, pulpal damage (inflammation) and tissue necrosis.

Clinical Features

- Localized chiefly to the pulpal ends of irritated dentinal tubules.
- Sensitive to thermal changes, especially to cold.
- Pain disappears upon removal of the thermal irritant.
- Lower pain threshold (responds to electric pulp testing at lower levels of current).
- Pain does not occur without stimulation.

Histologic Features

- Dilatation of pulp vessels.
- Extravasations of RBC's or some diapedesis of WBC's.

Treatment

- Irritant should be removed before the pulp is severely damaged.

(A) Acute Pulpitis

Clinical Features

- Frequent immediate sequelae of focal reversible pulpitis.
- Sharp and severe pain upon thermal stimulation and pain continues even after the stimulus is removed, in initial stages.
- Pain may be spontaneous or continuous may be increased when lying down.
- Tooth responds to electric pulp testing at lower levels during the early stage.
- In the later stages, pain increases in intensity, felt as a throbbing pressure after keeping the patient awake at night. Heat increases pain and cold may give some relief. Tooth responds to electric pulp testing at higher levels of current. If pulpal drainage occurs, symptoms subside.
- Until the inflammation or necrosis extends beyond the root apex, the tooth is not sensitive to percussion.

Histologic Features

- Early acute pulpitis.
- Continued vascular dilatation.

- Accumulation of edema fluid in the connective tissue surrounding tiny blood vessels.
- Pavementing of polymorphonuclear leukocytes.
- Destruction of odontoblasts.
- In case of abscess formation, it appears as a small void surrounded by dense band of leukocytes.
- Later stages, large numbers of bacteria are present.

Treatment and Prognosis

- Very early stages pulpotomy is done.
- Later stage root canal treatment is done.

(B) Chronic Pulpitis

- Chronic pulpitis may arise through either quiescence of an acute pulpitis or as a chronic type of disease from the onset.

Clinical Features

- Mild, dull intermittent pain.
- Due to degeneration of nerve tissue in the affected pulp, the threshold for stimulation by electric pulp testing is often increased.
- Pulp may become totally necrotic without pain.

Histologic Features

- Infiltration of pulp tissue by varying numbers of mononuclear cells, chiefly lymphocytes and plasma cells.
- Fibroblastic activity is evident.
- Prominent capillaries are seen.
- Bundles of collagen fibers are present.
- If the pulp attempts to wall off the infection through deposit of collagen about the inflamed area, it resembles the formation of granulation tissue and when this occurs on the surface of the pulp tissue with a wide open exposure, it is termed as ulcerative pulpitis.

Treatment and Prognosis

- Either root canal therapy or extraction.

CHRONIC HYPERPLASTIC PULPITIS

(Pulp polyp)

- Occurs either as a chronic lesion from the onset or as a chronic stage of a previous acute pulpitis.
- It is essentially an excessive, exuberant proliferation of chronically inflamed pulp tissue.

Clinical Features

- Occurs in children and young adults who have large exposures of the pulp in which the entire dentinal roof is missing.
- Appears as a pinkish red globule of tissue protruding from the pulp chamber often filling the entire cavity.
- Since the hyperplastic tissue contains few nerves, it is insensitive to manipulation.
- Mechanical irritation and bacterial invasion result in a level of chronic inflammation that produces hyperplastic granulation tissue that extrudes from the chamber.
- Commonly affected teeth are the deciduous molars and the first permanent molars which have an excellent blood supply because of large root apex along with a high tissue resistance and reactivity in young persons.
- Because of open apex, there is reduced chance of pulpal necrosis secondary to venous compression.

Histologic Features

- The hyperplastic tissue is basically granulation tissue made up of delicate connective tissue fibers interspersed with variable numbers of small capillaries
- Inflammatory cell infiltration, chiefly lymphocytes and plasma cells along with polymorphonuclear leukocytes is commonly found.
- The granulation tissue commonly becomes epithelized which is of stratified squamous type and resembles the oral mucosa.
- These epithelial cells may be either, desquamated cells carried to the surface of pulp by saliva (or)

Treatment and Prognosis

- Extraction or pulp extirpation

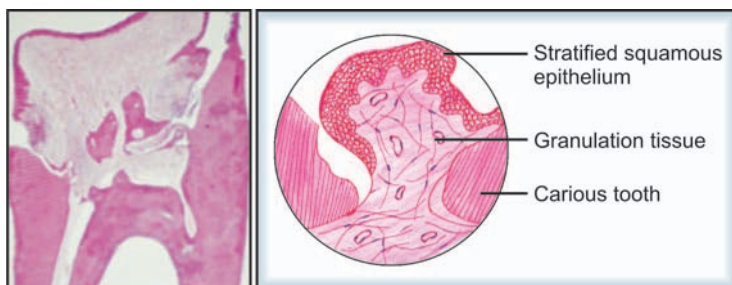
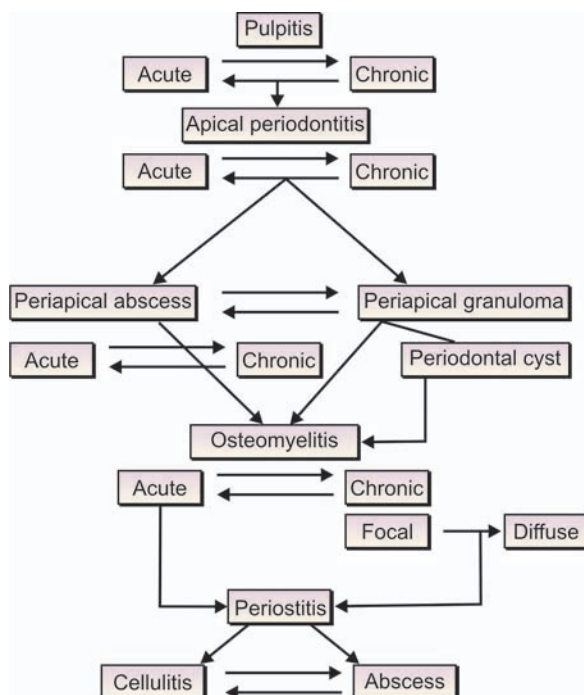


Fig. 3.1: Pulp polyp (chronic hyperplastic pulpitis)

DISEASES OF THE PERIAPICAL TISSUES

- Once infection becomes established in the dental pulp, it spreads through the root canals into the periapical region. These periapical do not represent individual distinct entities, there is a transformation from one type of lesion into another type.



PERIAPICAL GRANULOMA

(Apical periodontitis)

- The Periapical granuloma is a localized mass of chronic granulation tissue formed in response to the infection at the apex of a non-vital tooth. Formation of apical inflammatory reaction represents a defensive reaction secondary to the presence of bacteria in the root canal with spread of related toxic products into the apical zone.
- May arise following quiescence of periapical abscess or as the initial periapical pathosis.
- Lesions are not static and may transform into periapical cysts or may demonstrate acute exacerbation with abscess formation.

Clinical Features

- Mostly asymptomatic.
- In acute exacerbation noticeable sensitivity of involved tooth to percussion and pain may occur.
- Does not respond to electric or thermal pulp tests.
- No perforation of overlying bone and oral mucosa.

Radiographic Findings

- Appears as a radiolucent area of variable size attached to the root apex.
- A thin radiopaque line or zone of sclerotic bone may outline the lesion sometimes.
- Loss of apical lamina dura of affected tooth.

Histologic Features

- Periapical granulomas consists of inflamed granulation tissue surrounded by a fibrous connective wall.
- Demonstrates a variably dense lymphocytic infiltrate with neutrophils, plasma cells, histiocytes, mast cells and eosinophils.
- When numerous plasma cells are present, scattered eosinophilic globules of gamma globulin (**Russell bodies**) may be seen.
- Clusters of lightly basophilic particles (pyronine bodies) may also be present.
- Epithelial rests of malassez may be identified within the granulation tissue.

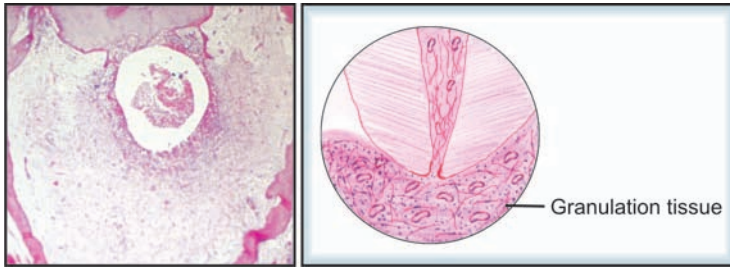


Fig. 3.2: Periapical granuloma (chronic apical periodontitis)

- Cholesterol crystal which appears as clear needle like spaces or clefts associated with multinucleated giant cells, areas of red blood cell extravasations with hemosiderin pigmentation may be present.
- Foam cells, which are phagocytosed when ingest lipid material, become collected as groups.
- The inflammation and locally increased vascularity of the tissue are associated with resorption of the adjacent supporting bone.

Treatment and Prognosis

- If the tooth can be maintained, root canal therapy can be performed with or without apicectomy.
- Non restorable teeth must be extracted, followed by curettage of all apical soft tissue.
- If left untreated, it may transform into an apical periodontal cyst through proliferation of epithelial cells in the area.

APICAL PERIODONTAL CYST

(Radicular cyst; periapical cyst; root end cyst)

- Epithelium at the apex of a nonvital tooth can be stimulated by inflammation to form a true epithelium lined cyst or periapical cyst. The source of epithelium is:
 - Cell rests of Malassez.
 - Crevicular epithelium.
 - Sinus lining.
 - Epithelial lining of fistulous tracts.

Pathogenesis

- Periapical cysts represent a fibrous connective tissue wall lined by stratified squamous epithelium with a lumen containing fluid and cellular debris. As the epithelium desquamates in the lumen, the protein content increases. Fluid enters the lumen in an attempt to equalize the osmotic pressure and slow enlargement occurs.

Clinical Features

- Asymptomatic unless there is an acute inflammatory exacerbation.
- Seldom painful or sensitive to percussion.
- Movement or mobility of adjacent tooth as the cyst enlarges.
- The tooth of origin does not respond to thermal and electric pulp testing.

Radiographic Findings

- The radiographic pattern is identical to that of periapical granuloma.
- Loss of lamina dura along the adjacent root and a rounded radiolucency encircles the affected tooth apex.
- Root resorption is common.
- Although periapical cysts attain a greater size than periapical granulomas, neither the size nor the shape of the lesion can be considered as a definitive diagnostic criteria.

Histologic Features

- Lining epithelium is usually stratified squamous, but in cases of lesion of maxillary sinus, the lining may be of pseudostratified ciliated columnar or respiratory type of epithelium.
- The epithelial lining may be missing over areas of intense inflammation.
- Presence of hyaline bodies which are tiny linear or arc shaped generally associated with the lining epithelium, that appear amorphous in structure, eosinophilic in reaction brittle in nature known as "Ruston bodies".

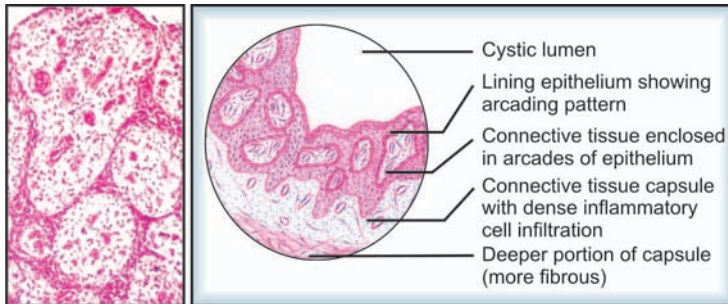


Fig. 3.3: Radicular cyst (periapical cyst, apical periodontal cyst, root end cyst)

- The connective tissue is composed of parallel bundle of collagen fibers, often with an inflammatory infiltrate containing lymphocytes along with neutrophils plasma cells, histiocytes, mast cells and eosinophils.
- Dystrophic calcification, cholesterol clefts with multinucleated giant cells, RBC's and areas of hemosiderin pigmentation may be present in the lumen wall or both.
- Histologically apical periodontal cyst is identical with periapical granuloma, except for the presence of epithelium - lined lumen.

Treatment and Prognosis

- Treatment is either through extraction of tooth and curettage or root canal therapy with apisectomy of the cystic lesion.
- If the cystic sac is badly fragmented, leaving epithelial remnants, a residual cyst may later develop.
- Epidermoid carcinoma may develop from the lining epithelium.

PERIAPICAL-ABSCESS

(Dento-alveolar abscess, alveolar abscess)

- The accumulation of acute inflammatory cells at the apex of a nonvital tooth is termed as periapical abscess.
- An acute inflammatory lesions with abscess formation may arise as the initial periapical pathosis (either caries or trauma) or from an acute exacerbation (phoenix abscess) of a chronic periapical inflammatory reaction.

Clinical Features

Acute periapical abscess

- Tooth is extremely painful.
- Tooth is slightly extruded from its socket.
- Regional lymphadenitis.
- Fever
- Malaise
- Headache
- Rapid extension to adjacent bone marrow spaces produces actual osteomyelitis.

Chronic periapical abscess

- No clinical features.
- A mild, well-circumscribed area of suppuration.

Radiographic Features

- Radiographically, abscesses may demonstrate a thickening of the apical periodontal ligament, an ill-defined radiolucency or both.
- Phoenix abscesses demonstrate the outline of the original chronic lesion with or without an associated ill-defined bone loss.

Histologic Features

- The area of suppuration is composed of a central area of disintegrating polymorphonuclear leukocytes along with inflammatory exudates, cellular debris, necrotic material, bacterial colonies or histiocytes.

Treatment and Prognosis

- Treatment involves drainage and elimination of the focus of infection which is done either by opening the pulp chamber or extracting the tooth.
If left untreated, periapical abscess may spread to result in
 - Osteomyelitis
 - Cellulitis
 - Bacteremia
 - Cavernous sinus thrombosis.

OSTEOMYELITIS

- Osteomyelitis can be an acute or chronic inflammatory process in the medullary spaces or cortical surfaces of bone that extends away from the initial site of involvement, usually a bacterial infection.

Predisposing Factors

- Chronic systemic diseases.
- Immunocompromised status.
- Disorders associated with decreased vascularity of bone.

(A) ACUTE SUPPURATIVE OSTEOMYELITIS

- Acute osteomyelitis exists when an acute inflammatory process spreads through the medullary spaces of the bone and there has been insufficient time for the body to react to the presence of the inflammatory infiltrate.
- It is a serious sequelae of periapical infection.

Clinical Features

- In Maxilla it is well localized to area of initial infection but in mandible it is more diffuse and widespread.
- In infants, osteomyelitis occurs either through hematogenous origin or a result of local oral infection. The affected infant will not survive normally.
- In adult the following are noted.
 - Severe pain and fever.
 - Regional lymphadenopathy
 - Leukocytosis
 - Significant sensitivity
 - Soft tissue swelling of the affected area
 - Paresthesia or anesthesia of lip
 - Loose sore teeth in area of involvement.

Radiographic Features

- Diffuse lytic changes in the bone begin to appear. Individual trabeculae become fuzzy and indistinct and radiolucent areas begin to appear.

Histologic Features

- The bone shows a loss of the osteocytes from their lacunae, peripheral resorption and bacterial colonization.
- Periphery of the bone and the haversian canals contain necrotic debris and an acute inflammatory infiltrate consisting of polymorphonuclear leukocytes.

Treatment and Prognosis

- Drainage should be established and maintained.
- Infection should be treated with antibiotics to prevent further spread of complications.
- When the intensity of the disease increases the bone that has lost its vitality begins to separate from the living bone. The separated fragment is called as “sequestrum”. When the sequestrum becomes surrounded by new living bone, it is called “involucrum”.
- Pathologic fracture may occur due to weakening of the jaw by the destructive process.

(B) CHRONIC SUPPURATIVE OSTEOMYELITIS

- Chronic osteomyelitis exists when the defensive response leads to the production of granulation tissue, which subsequently forms dense scar in an attempt to wall off the infected area. The encircled dead space acts as a reservoir of bacteria and antibiotics have greater difficulty in reaching the site.
- The clinical features are similar to those of acute osteomyelitis, except that all signs and symptoms are milder.
- Acute exacerbation of the chronic stage may occur and present all features of acute osteomyelitis.
- Chronic osteomyelitis is difficult to manage, because pockets of dead bone and organisms are protected from antibiotics by the surrounding wall of fibrous connective tissue, hence surgical intervention is mandatory.

(i) CHRONIC FOCAL SCLEROSING OSTEOMYELITIS

(Condensing osteitis)

- Localized areas of bone sclerosis associated with the apices of teeth with pulpitis or pulpal necrosis are termed condensing osteitis.
- Unusual reaction of bone to infection occurring in instances of extremely high tissue resistance or in cases of a low-grade infection.

Clinical Features

- Occurs more commonly in children and young adults.
- Most commonly involved tooth is the mandibular first molar with a large carious lesion.
- No signs or symptoms, other than mild pain associated with an infected pulp.

Radiographic Features

- The classic lesion consists of a localized, uniform zone of increased radio density adjacent to the apex of the tooth that exhibits a thickened periodontal ligament space.

Histologic Features

- Reveals a dense mass of bony trabeculae with little interstitial marrow tissue.

Treatment and Prognosis

- Associated tooth may be treated endodontically or extracted.
- A residual area of condensing osteitis which remains after resolution of the inflammatory focus is termed a bone scar.

Differential Diagnosis

- Benign cementoblastoma.
- Radiographically, the entire root outline is nearly always visible in case of condensing osteitis, as compared to benign cementoblastoma.

- Idiopathic osteosclerosis.
- Radiopacity is attached to the tooth apex in condensing osteitis.

(ii) CHRONIC DIFFUSE SCLEROSING OSTEOMYELITIS

- Analogous to the focal form of the disease. But mostly the route of entry is not through a carious lesion, but through diffuse periodontal disease.

Clinical Features

- Most common in older persons, especially in edentulous mandibular jaws or edentulous areas.
- Normally no clinical indication. In cases of acute exacerbation of the dormant chronic infection there is mild suppuration associated with formation of a fistula opening onto the mucosal surface. Patient may complain of vague pain and bad taste in the mouth.

Radiographic Features

- Radiopaque lesion may be extensive and bilateral. Because of the diffuse nature, the border between the sclerosis and the normal bone is often indistinct.
- Radiographically, may mimic the “cotton-wool” appearance of Paget’s disease.

Histologic Features

- Lesion shows dense, irregular trabeculae of bone with widely scattered haversian canals and little marrow tissue.
- The soft tissue between the individual trabeculae is fibrous and shows proliferating fibroblasts, small capillaries, lymphocytes, plasma cells and polymorphonuclear leukocytes.

Treatment and Prognosis

- Treated by resolution of the adjacent foci of chronic infection.

(iii) SCLEROTIC CEMENTAL MASSES

- Resembles chronic diffuse sclerosing osteomyelitis in many conditions.

Clinical Features

- Common in older black females.
- Multiple, symmetric lesions with pain, drainage, or localized expansion.

Radiographic Features

- Similar to diffuse sclerosis.

Histologic Features

- Sclerotic cemental masses may be differentiated from diffuse sclerosing osteomyelitis only microscopically.
- In sclerosing osteomyelitis, the tissue was essentially sclerotic bone, while in cemental masses, the tissue usually was cementum.

FLORID OSSEOUS DYSPLASIA

- Florid osseous dysplasia is an exuberant variant of osseous dysplasia which is an abnormal reaction of bone to irritation or stimulation.
- Resembles chronic diffuse sclerosing osteomyelitis and sclerotic cemental masses clinically, radiographically and histologically.
- One additional findings is the simultaneous occurrence of simple bone cysts.

(iv) CHRONIC OSTEOMYELITIS WITH PROLIFERATIVE PERIOSTITIS

(Garre's chronic nonsuppurative sclerosing osteitis; periostitis ossificans)

- Is a focal gross thickening of the periosteum of long bones, with peripheral reactive bone formation resulting from mild irritation or infection.
- Is essentially a periosteal osteosclerosis analogous to endosteal osteosclerosis of chronic sclerosing osteomyelitis.

Clinical Features

- Common in children and young adults especially in mandible.

- Greater opportunity for infection to enter the jaw bone because of the peculiar arrangement of the teeth situated in and protruding from the bone.
- Common cause is dental caries with periapical infection or overlying soft - tissue infection/cellulites.
- Toothache or pain in the jaw and a bony hard swelling on the outer surface of the jaw.

Radiographic Features

- Occlusal roentgenogram shows a focal overgrowth of bone on the outer surface of the cortex which may be described as a duplication of the cortical layer of bone which is smooth and well calcified.
- Radiopaque laminations of bone roughly parallel each other and underlying cortical surface.
- Radiolucent separations are present between the new bone and original cortex. Within the new bone, areas of small sequestra or osteolytic radiolucencies may be found.

Histologic Features

- The supracortical or subperiosteal mass is composed of much reactive new bone and osteoid tissue with osteoblasts bordering many of the trabeculae.
- The trabeculae are oriented perpendicular to the cortex.
- Connective tissue between trabeculae is fibrous with a diffuse or patchy sprinkling of lymphocytes and plasma cells.

Treatment and Prognosis

- Removal of the carious tooth is usually done.
- Biopsy may be done to confirm diagnosis.

Differential Diagnosis

- Periosteal new bone formation (neoperiostosis) may also occur in.

Caffey's disease (infantile cortical hyperostosis).

- Hypervitaminosis A
- Syphilis
- Leukemia
- Ewings sarcoma
- Metastatic neuroblastoma
- Fracture callus.

4

Diseases of Periodontium

Periodontism is defined as those tissues supporting and investing the tooth and consist of cementum, periodontal ligament, bone living the alveolus (socket) and that part of gingiva, facing the tooth periodontal diseases are pathological processes that affects the periodontium inflammation of periodontism can be manifested as gingivities or periodontism. Histologic studies of the periodontism seldom indicate the type of causing the disease or suggest a specific method of therapy.

Introduction

- It consists of four connective tissues, two hard and two soft.

Hard Tissues

- They include cementum and alveolar bone
- Cementum is a mineralized dental tissue covering the anatomic roots of human teeth.
- Alveolar bone is a part of maxilla and mandible that form and support the sockets of the teeth.

Soft Tissues

- They include periodontal ligament and gingiva
- Periodontal ligament is a fibrous connective tissue that connects

the two hard tissues of periodontium. It consists of cells and extracellular matrix.

- Gingiva is that part of oral mucous membrane that covers the alveolar processes of jaw and surrounds the neck of the teeth.
- The periodontium is affected by a variety of diseases that have been classified at the International Workshop for Classification of Periodontal Diseases.

Classification

- Gingival diseases.
- Plaque induced gingival diseases.
- Non-plaque induced gingival diseases.

Periodontitis

- Chronic periodontitis.
- Aggressive periodontitis.
- Periodontitis as a manifestation of systemic diseases.
- Necrotizing periodontal diseases.
- Necrotizing ulcerative gingivitis (NUG).
- Necrotizing ulcerative periodontitis (NUP).
- Abscesses of the periodontium.
- Periodontitis associated with endodontic lesions.
- Endodontic-periodontal lesions.
- Periodontal-endodontic lesions.
- Combined lesion.
- Developmental or acquired deformities and conditions.
- Localized tooth related factors.
- Mucogingival deformities and conditions on edentulous ridges.
- Mucogingival deformities and conditions around teeth.
- Occlusal trauma.

GINGIVAL DISEASES

- Gingivitis is the most common form.
- It is of two types:

(a) Plaque Induced Gingival Diseases

- Found on periodontium with no attachment loss or on periodontium with previous attachment loss that is stable and not progressing and is subdivided into four types.

(i) Gingivitis associated with dental plaque only

- It is a result of interaction between plaque microorganism and host's tissues and inflammatory cells and is altered by local factors, systemic factors or both.

(ii) Gingival diseases modified by systemic factors

- Endocrine changes associated with puberty, menstrual cycle, pregnancy and diabetes alter immune system which in turn alters gingival inflammatory response to plaque.
- Blood dyscrasias such as leukemia leads to gingival enlargement and bleeding with swollen, spongy gingival diseases.

(iii) Gingival diseases modified by medication

- Anticonvulsant drugs (phenytoin), immunosuppressive drugs (cyclosporine), calcium-channel blockers (sodium valproate) and increased use of oral contraceptives cause patient specific gingival enlargement.

(iv) Gingival diseases modified by malnutrition

- Scurvy causes red, swollen and bleeding gingiva.
- Affect immune function and host's ability to protect itself against the detrimental effects of cellular products.

(b) Non-plaque Induced Gingival Diseases

- They occur as oral manifestations of systemic conditions in developing countries, lower socioeconomic groups and immune-compromised individuals. It has seven origins.

(i) Gingival diseases of bacterial origin

- Increased prevalent is due to STD's (sexually transmitted diseases) like gonorrhea and syphilis.
- Streptococcal species causes gingivostomatitis, an acute infection with fever, malaise and pain associated with acutely inflamed, red, swollen and bleeding gingiva.

(ii) Gingival diseases of viral origin

- These are caused by DNA and RNA (Herpes) viruses.

(iii) Gingival diseases of fungal origin

- They are rare and seen in immunocompromised individuals or in people whose normal oral flora has been disturbed by long-term use of broad spectrum antibiotics.
- Most common is Candidiasis (*Candida albicans*).
- HIV-associated gingivitis or linear gingival erythema occurs in HIV patients manifesting as erythema of attached gingiva.

(iv) Gingival diseases of genetic origin

- Most common is hereditary gingival fibromatosis.
- Gingival enlargement may completely cover teeth and delay eruption.

(v) Gingival manifestations of systemic conditions

- They appear as desquamative lesions or ulceration of the gingiva or both.
- Restorative materials, tooth pastes, mouthwashes and foods may sometimes cause allergic reactions in gingiva.

(vi) Traumatic lesions

- They may be factorial (toothbrush trauma), iatrogenic (restorative care) or accidental (minor burns through hot edibles).

(vii) Foreign body reactions

- These occur by introduction of foreign materials into gingival connective tissue leading to localized inflammatory conditions, e.g. amalgam introduction into gingiva while restoring a tooth.

PERIODONTITIS

- Inflammatory disease of supporting tissues of teeth caused by specific microorganisms; resulting in progressive destruction of periodontal ligament and bone with pocket formation, recession, or both is called periodontitis.
- Differs from gingivitis by presence of clinically detectable, continuous or episodic attachment loss.
- Continued bleeding on probing at sequential visits is a good indicator of this disease.
- Classifications of periodontitis are based on three factors, i.e. age of onset, rate of disease progression and present of alterations in host defenses.

1989 Classification by AAP

- Adult periodontitis.
- Early onset periodontitis (Juvenile, prepubertal or rapidly progressive).
- Periodontitis associated with systemic diseases.
- Necrotizing ulcerative periodontitis (similar to gingivitis but with clinical attachment loss).
- Refractory periodontitis.

1993 Classification by European workshops

- Adult periodontitis.
 - Early onset periodontitis.
 - Necrotizing periodontitis.
- The clinical and etiologic manifestations of different diseases outlined in 1989 and 1993 classification were not consistently observed and did not always fit models. Hence in 1999 AAP presented another classification.

1999 Classification by AAP

- Chronic periodontitis.
- Aggressive periodontitis.
- Periodontitis as a manifestation of systemic diseases.

Chronic Periodontitis

- Most common form of periodontitis.
- Similar to adult periodontitis, i.e. it occurs mostly in adults but it can also occur in children.
- It is associated with accumulation of plaque and calculus and has slow to moderate rate of disease progression.
- The rate of disease progression is sometimes rapid due to factors like systemic diseases (HIV), local factors and environmental factors (cigarette smoking).

Aggressive Periodontitis

- It is similar to early onset periodontitis.
- Associated with rapid attachment loss and bone destruction in an otherwise healthy individual and also in case of absence of familial history.

- The following characteristics are common but not universal in this disease.
 - Diseased site infected with *Actinobacillus actinomycetemcomitans*.
 - Abnormality in phagocyte function.
 - Self arresting disease progression.
 - Both chronic and aggressive periodontitis can be subclassified as.
 - Localized form : <30 percent of sites involved.
 - Generalized form : >30 percent of sites involved.
- OR
- Slight: 1 to 2 mm of clinical attachment loss.
 - Moderate : 3 to 4 mm of clinical attachment loss.
 - Severe: 5 mm of clinical attachment loss.

Periodontitis as a manifestation of systemic disease

- Systemic diseases alter the host defense mechanism and lead to periodontitis.

Systemic diseases involved

- Hematologic disorders like acquired neutropenia and leukemias.
- Genetic disorders like Down syndrome, Papillon Lefèvre syndrome and familial and cyclic neutropenia.

NECROTIZING PERIODONTAL DISEASES

- It manifests as marginal gingivitis covered by yellowish white or grayish slough or pseudomembrane, blunting or catering of papillae, bleeding on provocation or spontaneous bleeding.
- This is accompanied by fever, malaise and lymphadenopathy.

It is of two types but tissue necrosis is primary clinical feature of both types

(a) Necrotizing Ulcerative Gingivitis (NUG)

- Its features are bacterial etiology, necrotic lesion and predisposing factors are such as stress, smoking, immuno-suppression and malnutrition seen in developing countries only.
- Clinically seen as acute lesion responding well to antimicrobial therapy combined with professional plaque and calculus removal and improved oral hygiene.

(b) Necrotizing Ulcerative Periodontitis (NUP)

- Its clinical features are similar to NUG with a few exceptions.
- Involves loss of clinical attachment and bone.
- In HIV infected patients it manifests as local ulceration and necrosis of gingival tissue with rapid destruction of underlying bone, spontaneous bleeding and severe pain.

ABSCESSSES OF THE PERIODONTIUM

- A periodontal abscess is a localized purulent infection of periodontal tissue.
- It is classified by its tissue of origin into three types:
 - Gingival abscess.
 - Periodontal abscess.
 - Pericoronal abscess.

PERIODONTITIS ASSOCIATION WITH ENDODONTIC LESIONS

- It involves periodontium as well as pulp.
- Classification is based on the disease process sequence.

(a) Endodontic-Periodontal Lesions

- Here pulpal necrosis precedes periodontal changes.
- Periapical lesion originating in pulpal infection and necrosis may drain to oral cavity through periodontal ligament or accessory canals.
- Consequently there will be destruction of periodontal ligament and alveolar bone, clinically presenting as periodontal pocket extending to apex.

(b) Periodontal-Endodontic Lesions

- Bacterial infection from a periodontal pocket associated with attachment loss and root exposure may spread through accessory canals or apical foramen (in case of advanced periodontal diseases only) to pulp, causing pulpal necrosis.

(c) Combined Lesions

- They occur when pulpal necrosis and periapical lesion occur on a tooth that is also periodontally involved.
- Both combine to form an intrabony effect.
- In such cases, endodontic infection should be controlled before managing periodontal lesions.

DEVELOPMENTAL OR ACQUIRED DEFORMITIES AND CONDITIONS

They are five main causes:

(a) Localized tooth related factors.

- They contribute to initiation and progression of periodontal diseases through enhancement of plaque accumulation or prevention of effective plaque removal by normal oral hygiene measures.

It is subdivided into

i. Tooth anatomic factors.

- They are associated with malformations of tooth development and tooth location.

ii. Dental restorations or appliances.

- Restorations may impinge on biologic width by being placed deep in the sulcus or within junctional epithelium leading to loss of clinical attachment and bone with apical migration of the junctional epithelium.

(b) Root fractures.

- Root fractures originating coronal to clinical attachment and exposed to oral environment may lead to periodontal involvement through apical migration of plaque along fracture.

(c) Cemental tears and cervical root resorption.

- They may lead to periodontal destruction when the lesion communicates with oral cavity and allows bacteria to migrate subgingivally.

(d) Mucogingival deformities and conditions around teeth.

- i. Mucogingival deformity refers to significant departure from normal shape of gingiva and alveolar mucosa.

These include conditions like:

- Gingival/soft tissue recession.
 - Lack of keratinized gingiva.
 - Decreased vestibular defects.
 - Aberrant frenum or muscle position.
 - Gingival excess.
 - Pseudopocket.
 - Inconsistent gingival margin.
 - Excessive gingival margin.
 - Gingival enlargement.
 - Abnormal color.
- ii. Mucogingival deformities and conditions on edentulous ridges.

These include:

- Vertical and horizontal ridge deficiency.
- Lack of gingiva or keratinized tissue.
- Gingival or soft tissue enlargement.
- Aberrant frenum or muscle position.
- Decreased vestibular depth.
- Abnormal color.
- These require corrective surgery to restore form and function before prosthetic replacement of missing teeth or implant placement.

(e) Occlusal trauma

These include:

- Primary occlusal trauma.
- Secondary occlusal trauma.

Physical and Chemical Injuries of the Oral Cavity

PHYSICAL INJURIES OF THE TEETH

BRUXISM

(Night-grinding; Bruxomania)

Definition

Habitual grinding of teeth, either during sleep or as an unconscious habit during working hours.

Etiology

- Local factors:
 - Occlusal disturbances.
- Systemic factors:
 - Gastrointestinal disturbances.
 - Nutritional deficiencies.
 - Endocrine disturbances.
 - Hereditary background.
- Psychologic factors:
 - Tension.
 - Mental illnesses.

- Occupational factors:
 - Watchmakers.
 - Athletes.

Clinical Features

- Grinding or clenching motions associated with creating noise.
- Severe wearing or attrition of the teeth.
- Loss of integrity of the periodontal structures.
- Loosening or drifting of teeth.
- Gingival recession with alveolar bone loss.
- Injury to temporomandibular joint due to continuous clenching of teeth.
- Hypertrophy of masticatory muscles.
- Facial pain.
- Headache.

Treatment

- Correct the underlying cause.
- Removable splints to be worn at night to immobilize jaw or guide the movement.

FRACTURES OF TEETH

Causes

- Trauma.
- Tooth with large restoration.
- Internal resorption.
- Endodontically treated tooth.
- Dentinogenesis imperfecta.

Ellis Classification of Fracture of Anterior Teeth

- Class I: Simple fracture of crown, involving little or no dentin.
- Class II: Extensive fracture of crown, involving dentin, not pulp.
- Class III: Extensive fracture of crown, involving dentin and pulp.
- Class IV: Tooth is non vital.
- Class V: Teeth lost due to trauma.
- Class VI: Fracture of root with or without loss of crown structures.

- Class VII: Displacement of a tooth, without fracture of crown or root.
- Class VIII: Fracture of crown enmasse and its replacement.
- Class IX: Injuries to deciduous teeth.

Clinical Features

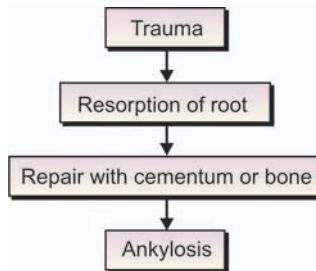
- Occurs at any age children are especially prone
- Boys are more frequently involved than girls.

TOOTH ANKYLOSIS

- Union of tooth with bone.
- It is a very rare condition.

Causes

- Trauma



- Periapical inflammation.
- Following root canal treatment, if the apical periodontal ligament is irritated or damaged.

Clinical Features

- Dull, muffled sound on percussion.
- Difficult to extract the tooth.

Radiographic Features

- Loss of normal thin radiolucent line that represents the periodontal ligament.

Treatment

- No treatment for ankylosis.
- Any infection present should be treated.

PHYSICAL INJURIES OF THE BONE

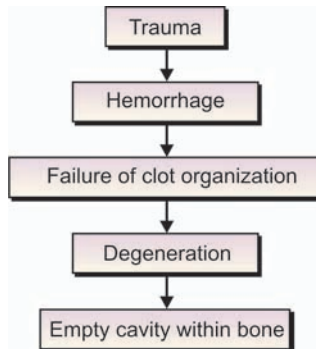
TRAUMATIC CYST

(Solitary bone cyst, idiopathic bone cavity, simple bone cyst)

- Unusual lesions.
- Pseudocyst, since the intrabony cavities are not lined by epithelium.

Etiology

- Trauma



- Inadequate venous drainage causes inability of the interstitial fluid to exit bone.
- Local disturbance in bone growth.
- Ischemic marrow necrosis.
- Alteration in bone metabolism causes osteolysis.
- Cystic degeneration of bone tumors.
- End result of a low grade chronic infection.
- Disturbed circulation caused by trauma creating an unequal balance of osteoclasts and bone repair.

Clinical Features

- Frequently affects young persons.
- Males commonly affected than females.

- Common site is posterior portion of mandible.
- It is discovered during routine radiographic examination.
- Involved teeth remains vital.
- Bone expansion is rare.
- On surgical opening, it contains shreds of necrotic blood clot, fragments of fibrous connective tissue or nothing.

Radiological Features

- Ovoid radiolucent defect above mandibular canal.
- Scalloped outline between roots of tooth.

Histologic Features

- Thin connective tissue membrane lining the cavity. Rarely in addition to this extensive osteophytic reaction on the outer side of the cortical plate.

Treatment

- Open into the cavity and enucleate lining, re-establish bleeding into the lesion which enhances healing.

FOCAL OSTEOPOROTIC BONE MARROW DEFECT

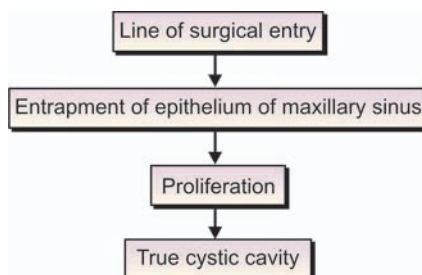
- Common in mandibular molar area. Common in female.
- Seen as ovoid radiolucent defect in the bone marrow.
- Most common in edentulous areas as a result of failure of normal bone regeneration after tooth extraction.

SURGICAL CILIATED CYST OF MAXILLA

(Implantation cyst)

- It develops after surgical entry into the maxillary sinus.

Pathogenesis



Clinical Features

- Tenderness or discomfort in the maxilla.
- Extraoral or intraoral swelling.

Radiographic Feature

- Well-defined radiolucent area in the maxillary sinus.

Histologic Features

- Cyst is lined by pseudostratified ciliated columnar epithelium.

Treatment

- Enucleation of the cyst.

PHYSICAL INJURIES OF SOFT TISSUES

TRAUMATIC ULCER

Causes

- Biting the mucosa
- Denture irritation
- Toothbrush injury
- Trauma from sharp tooth.
- Tooth brushing and cotton roll injury.

Common Sites

- Lateral border of tongue
- Buccal mucosa
- Palate
- Lips.

FACTITIAL INJURIES

- Accidentally self induced injuries on the basis of habit with a frequent psychogenic background.

Causes

- Lip biting
- Cheek biting.

DENTURE INJURIES

- Sore spots.
- Denture stomatitis.
- Epulis fissuratum.
- Palatal papillomatosis.
- Allergy.

TRAUMATIC ULCER

(Sore spots)

Causes

- Over extension of the flanges.
- Bony spicules under the denture.
- High points on the denture.

Clinical Features

- Small, painful lesions surrounded by erythematous halo, covered by necrotic membrane.

Histologic Features

- Disruption in epithelial continuity.
- Inflammatory cell infiltration.
- Dilation of capillaries.

Treatment

- Correction of underlying cause.

DENTURE SORE MOUTH

(Denture stomatitis)

Causes

- *Candida albicans* infection/chronic atrophic candidiasis.
- Poor denture cleanliness.
- Continual wearing of denture.
- Dietary and systemic alterations.

Clinical Features

- Red, swollen and painful mucosa beneath the denture.
- Area of pinpoint hyperemia.
- Severe burning sensation.

Treatment

- Nystatin tablets - 50,000 units were allowed to dissolve in the mouth three times a day for 14 days.
- Replacing denture.
- Rebasing with tissue conditioners.

INFLAMMATORY FIBROUS HYPERPLASIA

(Epulis fissuratum)

Cause

- Chronically ill fitting denture.

Clinical Features

- Excessive fold of tissue at mucolabial and mucobuccal area, near flanges.
- Ulceration in the base of the fold.
- Firm on palpation. Leaf like projection seen.
- Occurs in middle age with female predilection.

Histologic Features

- Fibrous connective tissue covered by a layer of stratified squamous epithelium.
- Pseudoepitheliomatous hyperplasia.
- Connective tissue composed of coarse bundles of collagen fibers, fibroblasts and blood vessels.
- Surface epithelium shows mucopolysaccharide keratin dystrophy or plasma pooling. It is also seen in other conditions where epithelium is irritated.
- It consist of eosinophilic pools of material in superficial spinous layer.

Treatment

- Surgical excision.
- Replacement of new dentures.

INFLAMMATORY PAPILLARY HYPERPLASIA

(Palatal papillomatosis)

- It is a form of inflammatory hyperplasia.
- Commonly affects palatal mucosa.
- Possible cause is ill fitting dentures and constant wearing of dentures.

Clinical Features

- Numerous red, edematous papillary projections involving hard palate and sometimes extend to the alveolar mucosa.

Histologic Features

- Numerous projections or papillae lined by stratified squamous epithelium.
- Pseudoepitheliomatous hyperplasia.
- Connective tissue shows inflammatory cell infiltration.

Treatment

- Surgical excision.
- Construction of new dentures.

ALLERGY TO DENTURES

- Reaction to methyl methacrylate, cobalt-chromium alloy used as base materials. Nickel in cast partial denture and sulfur in the base material.
- Generalized inflammation.
- Confirmed by patch test.

PERLECHE

(Angular cheilitis, angular cheilosis)

Etiology

- *Candida albicans*.
- Decreased vertical dimension in edentulous patients and patients with artificial dentures.
- In this condition a fold is produced at the corners of the mouth, saliva tends to collect, skin becomes fissured and secondarily infected.
- Riboflavin deficiency.

Clinical Features

- Occurs in young children and adults.
- Characterized by dryness, burning sensation at the corners of the mouth.
- Deep fissures or cracks which appear ulcerated and covered superficially by exudative crust.

Treatment

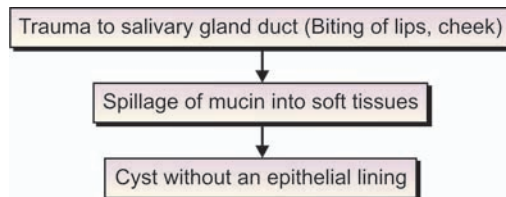
- Treat the primary cause and then the treatment for secondary infection.

MUCOCELE

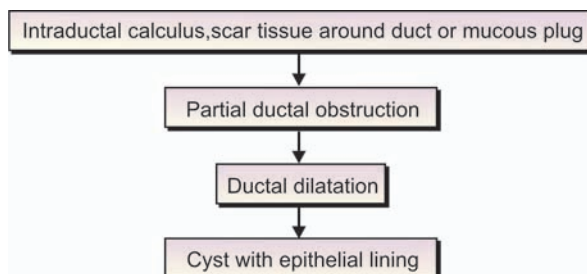
- Lesion involving minor salivary glands and their ducts.

Etiology

- **Mucous extravasation cyst : False cyst**



- **Mucous retention cyst : True cyst**



Clinical Features

- *Site:* Lower lip, floor of mouth, buccal mucosa, retromolar area, soft palate.
- *Age:* Children and young adults.
- Dome shaped fluctuant mucosal swelling.
- Superficial mucocele is bluish in color. In deeper mucocele, color and surface appearance resembles normal mucosa.
- History of recurrent swelling with periodic rupture.

Histologic Features

- Area of spilled mucin in connective tissue surrounded by granulation tissue.
- Lumen of the cyst is filled with an eosinophilic coagulum.
- Connective tissue shows infiltration of macrophages (foamy histiocytes), lymphocytes and plasma cells.
- Adjacent minor salivary gland shows sialadenitis.
- The epithelial lining of retention cyst is cuboidal, columnar, squamous epithelium.

Treatment

- Excise along with associated minor salivary gland.

RANULA

- Rana means frog, similar to frog's belly.
- Retention mucocele in floor of mouth.
- Associated with ducts of sublingual and submandibular gland.

Clinical Features

- Painless mass, seen in floor of mouth.
- Plunging type of ranula herniates through the mylohyoid muscle, seen as swelling in neck.

Histologic Features

- Microscopic appearance is similar to that of the smaller mucocele except that a definite epithelial lining is sometimes present.

Treatment

- Excision of sublingual or submandibular gland to prevent recurrence.

SIALOLITHIASIS

(Salivary duct calculus)

- Calcerous concentrations in the salivary ducts or glands.
- Formed by deposition of calcium salts around a central nidus consist of desquamated epithelial cells, bacteria, foreign bodies or products of bacterial decomposition.

Reasons

- High mucin content causes adherence to foreign particles.
- Long and irregular course of duct.

Clinical Features

- Submandibular gland duct is commonly involved.
- Minor glands of upper lip, buccal mucosa affected.
- Middle aged adults are affected.
- Complains of moderate to severe pain just before meals.

Compositions

- Calcium phosphate, calcium carbonate, water, organic salts.

Radiographic Features

- Radiopaque mass, ovoid in shape.

Treatment

- Small calculus is removed by manipulation.
- In case of large calculus, gland is excised calculus.

EFFECTS OF RADIATION

(a) Effects of X-ray on Cells

- Inactivated enzymes.
- Denaturation and coagulation of cellular proteins.



- Toxic effect on protein breakdown product.

Most radiosensitive cells are:

- Lymphocytes and lymphoblasts.
- Myeloblastic and erythroblastic cells (bone marrow).
- Epithelium of intestine and stomach
- Germ cells
- Mitotic phase cells.

Effects of radiation depends on

- Source
- Total amount
- Exposure time
- Area of tissue
- Type of filtration used.

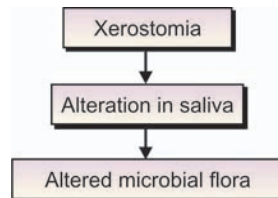
Effects on Oral Mucosa

- *Mucositis*: Initially, soreness and swelling. Intense erythema develops after 48 -72 hours. Later ulceration and denudation occurs. Damage to taste buds. Remission → Termination of therapy.

Effects on Salivary Glands



- Histologically, there is decrease in zymogen granules, edema, inflammatory cell infiltration in connective tissue.



Effects on Teeth

- Radiation caries begins at cervical portion of the tooth.
- Brittle teeth.
- Radiation caries is prevented by daily application of 1 percent sodium fluoride gel.
- Dental caries.

- In developing teeth: cessation of odontogenesis leads to anodontia.

(e) Effects on Bone

- Osteoblasts are radiosensitive.
- Decrease in vitality of bone.
- Alteration in bone remodeling.
- Slow and poor healing of extractions sockets.
- Inflammatory response is decreased, so bone fails to react normally to infections.

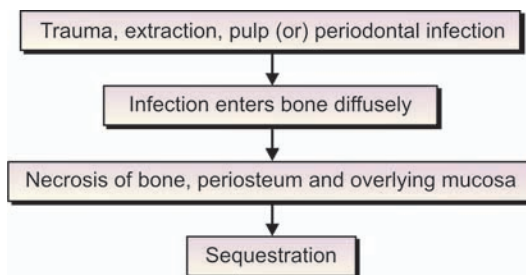
OSTEORADIONECROSIS

- It is a pathologic process which follows heavy radiation of bone.
- Characterized by chronic painful infection and necrosis accompanied by sequestration and sometimes permanent deformity.

Clinical Features

- Mandible commonly affected than maxilla.
- Intense pain.

Pathogenesis



Predisposing Factors

- High dose of radiation to bone.
- Irradiating an improperly healed surgical area.

- Surgery in an irradiated area.
- Irradiating lesions close to bone.

Patient Factors

- Poor oral hygiene.
- Poor patient cooperation.
- Indiscriminate use of prosthetic appliances.
- Nutritional deficiencies.

CERVICOFACIAL EMPHYSEMA

- Emphysema—Swelling due to air or gas in the connective tissue.

Causes

- Extraction
- Use of compressed air into root canal.
- Fracture of middle third of face.

Clinical Features

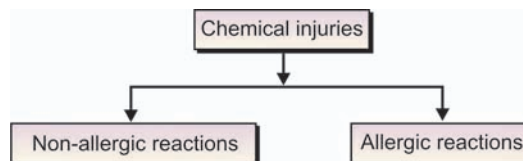
- Unilateral, painful, rapidly expanding swelling of face and neck.
- Difficulty in breathing.

Treatment

- Resolves in a week.
- If secondary bacterial infection is present, administer antibiotics.

Complication

- Venous air embolism



1. Drugs / chemicals - Locally
2. Drugs / chemicals - Systemically

1. Angioneurotic edema
2. Drug allergy (or) stomatitis medicamentosa
3. Contact stomatitis (or) stomatitis venenata

NON-ALLERGIC REACTIONS TO DRUGS AND CHEMICALS USED LOCALLY

Aspirin Burn

- Separation and sloughing of epithelium.
- Blanched mucosa.

Sodium Perborate

- Used in mouthwashes, dentifrices.
- Causes erythema and sloughing.

Phenol, Silver Nitrate, Volatile Oils

- Used as cavity sterilizing agent.
- Causes burning sensation of mucosa.

NON - ALLERGIC REACTIONS TO DRUGS USED SYSTEMICALLY

Bismuth

- Used in the treatment of skin diseases.
- Manifests as thin, blue - black line in marginal gingiva or papilla.
- Hydrogen sulfide produced by bacterial degradation of organic material or food debris, reacts with bismuth in tissues and produce bisulfide granules, which causes discoloration.
- Burning sensation, metallic taste.

Treatment

- Bleaching of bismuth lined by hydrogen peroxide.
- Discontinue bismuth usage.

Dilantin Sodium

- Gingival hyperplasia starts at interdental papilla, 2-3 months after therapy.
- gingival surface.

Histopathology

- Increased thickness of epithelium.

- Test tube retepegs.
- Bundles of collagen fibers with fibroblasts.

Treatment

- Surgical excision.
- Discontinuing use of the drug.

Lead Poisoning

(Plumbism)

- Occupational hazard.
- Inhalation of vapors.
- Paints.

Clinical Features

- Gastrointestinal disturbances.
- Anemia.
- Peripheral neuritis.

Oral Manifestations

- “Lead Line” → Gray/bluish-black line in gingiva.
- Ulcerative stomatitis.
- Increased salivation (ptyalism).

Mercury

- Gastrointestinal disturbances. Tremor on lips and nephritis.

Oral Manifestations

- Ptyalism
- Metallic taste
- Enlarged salivary glands
- Hyperemic and swollen gingiva
- Ulceration
- Gingival pigmentation
- Exfoliation of teeth.

Acrodynia

(Pink’s disease/Swift’s disease)

- Chronic exposure to Mercury in infants and children.

Source

- Ointments, disinfectants.

Clinical Features

- Affects young infants (< 2 years).
- Skin of extremities and cheek resembles “raw beef”.
- Pruritic maculopapular rash.
- Increased sweating.

Oral Manifestations

- Ptyalism
- Bruxism
- Gingival ulceration.

Treatment

- BAL (British Anti - Lewisite - Dimercaprol).

Silver

(Argyria, Argyrosis)

- Exposure
 - Occupational
 - Use of silver nitrate or other silver compounds.

Effects

- Pigmentation of skin, oral mucosa diffusely (Bluish-gray) due to reduction of silver.

AMALGAM TATTOO

- During filling, silver particles get embedded in gingiva.
- During removal of restoration, particles enter lacerated mucosa.
- During tooth extraction, silver particles enter surgical wound during retrograde filling.

Sites

- Gingiva (most common), buccal mucosa, alveolar mucosa.

Histopathology

- Black/brown granules along collagen fibers and blood vessels.
- Tissue shows foreign body reaction or no response.

Tetracycline

- It forms a complex with calcium salts in teeth which occurs discoloration (Yellowish /brownish gray).

Factors

- Time of exposure
- Type of drug
- Dosage.

Chemotherapeutic Drugs

- Ulceration - due to neutropenia.
- Hemorrhage - due to thrombocytopenia.
- Infections - candida, herpes simplex.
- Pigmentation of oral mucosa.

ALLERGIC REACTIONS TO DRUGS AND CHEMICALS

“Allergy”

- Encompasses the hypersensitive state acquired by exposure to a specific material and the altered capacity of the living organism to react upon re-exposure to this allergen.
- Involves antigen antibody reaction.

ANGIOEDEMA/ANGIONEURETIC EDEMA/QUINCKE'S DISEASE

- Diffuse edematous swelling of soft tissue.
- Allergic reaction occurs immediately.

Etiology

- Basic mechanism is increase in vascular permeability.

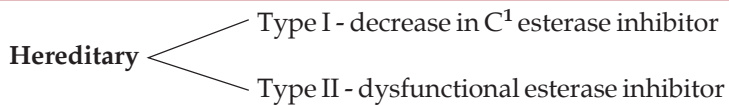
MAST CELL DEGRANULATION

Occurs due to

- Foods
 - Drugs
 - Cosmetics
 - Topical medications
 - Rubber dams.
- It is an IgE mediated hypersensitivity.

COMPLEMENT PATHWAY ACTIVATION

- Hereditary
- Acquired



- C¹ esterase inhibitor - prevents transformation of C¹ to C¹ esterase.
- Lack of C¹ inhibitor causes increase in C¹ esterase. C leaves C⁴ and C² and produce kinins and other anaphylatoxin → Increased vascular permeability → Angioedema.

ACQUIRED

- Lymphoproliferative disorders and in autoimmune condition.

Clinical Features

- Diffuse swelling around lips, chin, eyes.
- Puffy lips, shut eyes.
- Erythema and pruritis.
- Lasts for 24 - 36 hours.
- Edema of glottis, leads to suffocation.

Treatment

- Antihistaminics (corticosteroids).
- C¹-INH concentrate and esterase inhibiting drugs - Tranexamic acid.
- Androgens (Danazol) - for hereditary and acquired forms which induces hepatic synthesis of C¹- INH.

STOMATITIS MEDICAMENTOSA

- It is the allergic reaction of oral mucosa to systemically administered medications.

Clinical Features

- Skin lesions with urticaria.

Oral Manifestations

- Erythema, ulceration.
- Vesicles / bullae.
- Ulceration and necrosis of gingiva which is similar to ANUG.
- Black hairy tongue.

Systemic Manifestations

- Arthralgia
- Lymphadenopathy
- Agranulocytosis
- Fixed drug eruption.

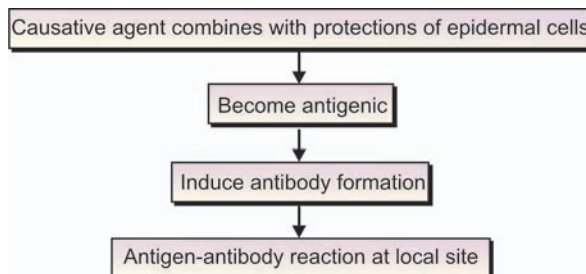
Treatment

- Antihistamines
- Corticosteroids.

CONTACT STOMATITIS / STOMATITIS VENENATA

- It occurs due to repeated contact with the causative agent.
- It manifest as lesions of skin or mucous membrane.

Mechanism



Causative Agents

Dental/Cosmetic preparations

- Dentifrices (flavoring agent)
- Mouthwashes
- Cough drops
- Chewing gums.

Dental materials

- Metal alloys.

Clinical Features

- Itching or burning at contact site.
- Erythema shiny appearance.
- Vesicles formation.

Treatment

- Discontinue agent.

Regressive Alteration of the Teeth

Regressive changes in the dental tissues include a variety of alterations that are not necessarily related either etiologically or pathogenetically. Some alterations are due to general aging process of the individual but some are due to injury to the tissues.

Regressive alterations of teeth are the group of retrogressive changes, which occur due to non-bacterial causes and results in wear and tear of the tooth structures with impairment of function.

ATTRITION

Definition

- Physiologic wear of tooth substance or restoration as a result of tooth-to-tooth contact during mastication or parafunctional habits.
- It is an age related process.
- It depends upon several factors such as diet quality, dentition, force of the masticatory muscles, chewing habits.

Types

- Physiological
- Pathological

(a) Physiological

- Maintains a constant pattern and it is proportionate to the age of the individual.
- Begins with wearing of the incisal edge of incisors, followed by palatal cusp of maxillary molars, buccal cusp of mandibular molars.
- Also occurs in the proximal surfaces of teeth in the contact point areas.

(b) Pathological

- Due to certain abnormalities in occlusion, chewing pattern or due to structural defects in the teeth.
- Does not maintain a consistent pattern.
- The amount of tooth loss is not proportionate to the age of the individual.

Causes

- Abnormal occlusion includes developmental (e.g. crowding/ malposed teeth) and acquired (e.g. extraction of teeth increases occlusal load on remaining teeth).
- Abnormal chewing habits, (e.g. bruxism, tobacco and betelnut chewing).
- Structural defects in teeth (Amelogenesis imperfecta, Dentinogenesis imperfecta).

Clinical Features

- Commonly seen in males.
- Well - polished facets on the tip of the cusps, incisal edges and on the proximal contact areas of the teeth.
- Severe attrition - complete wearing of enamel and flattening of the occlusal surface.
- Dentin exposure shows brown discoloration and hypersensitivity.
- Attrition in proximal surface leads to mesial migration of teeth in the arch.
- Even lead to fracture of cusps of teeth / restorations.

Treatment

- Correction of developmental abnormalities.
- Correction of parafunctional chewing habits.
- Protection of tooth by crowns where structural defects exist.
- Construction of occlusal guard if bruxism habit is persisting.

ABRASION

Definition

- Pathological wearing of dental tissues by friction with the foreign substances.

Causes

- Toothbrush Abrasion.
 - Faulty tooth brushing technique causes cervical abrasion.
 - Abrasive dentifrice.
- Habitual Abrasion
 - Pipe smokers-Incisor edges of upper and lower anterior teeth abrasion.
- Improper habitual use of tooth pick/ dental floss - proximal surface abrasion.
- Occupational Abrasion
 - Hairdressers - grip hairpins between their teeth - Notching of incisal edges.
 - Carpenters - Small tools / nails between their teeth - Notching of incisal edges.
 - Tailors and shoe makers grip needles between their teeth, so notching of incisal edges are seen.
- Prosthetic Appliances
 - Faulty clasp design in RPD causes cervical abrasion.

Clinical Features

- Type and severity of abrasion depends upon duration, type of faulty habit adopted by the person.
- Maxillary teeth are commonly affected than mandibular teeth.
- "V" shaped or wedge shaped groove on the tooth with sharp angles and highly polished dentin surfaces.

- Severe abrasion causes exposure of dentinal tubules and hypersensitivity.

Treatment

- Avoid abnormal brushing habits.
- Restorative treatment should be done to treat already abraded tooth.

EROSION

Definition

- Progressive irreversible loss of hard dental tissues by chemical processes. Dissolution of the mineralized tooth structure occurs upon contact with acids either from extrinsic or intrinsic sources.

(a) Extrinsic Factors

- Acidic foods and beverages.
 - Regular consuming of fruit juices, carbonated soft drinks with low pH.
- Medications.
 - Vitamin C and hydrochloric acid preparations that are chewed or kept in mouth for a long-time.
- Occupational erosion.
 - Vapors of chromic acid, hydrochloric acid, sulphuric acid, and nitric acid in industrial electrolyte process.
 - Workers in manufacturing of lead acid batteries or sanitary cleansers.
 - Swimmers (pool water will be having increased concentration of acids).

(b) Intrinsic Factors

- In gastroesophageal reflux diseases, excessive vomiting occurs so erosion commonly occurs on the lingual or palatal surfaces of maxillary teeth.
- Chronic alcoholism associated with frequent vomiting.
- Patients with hyperthyroidism, erosion of teeth is three times higher than normal patients.

- Decrease salivary flow due to medication or disease, decrease buffering action of saliva causes increase erosion.

Clinical Features

- Commonest site of dental erosion is the gingival third of the labial surfaces of maxillary incisors.
- Chronic cases - proximal surfaces of the teeth are also involved.
- Shallow, broad, 'scooped out' concavities on the enamel with highly polished surfaces.
- Exposure of dentin causes hypersensitivity which triggers secondary dentin formation.

Treatment

- Identification and stoppage of the etiology (counseling in case of consuming excessive amount of carbonated beverages).
- Proper therapy for patients with chronic vomiting or GERD (Gastroesophageal reflux disease).

ABFRACTION

- It is a wedge shaped defect limited to the cervical area of the teeth and may closely resemble cervical abrasion or erosion.

Clinical Features

- It is diagnosed as a deep, narrow and V shaped (which don't allow the toothbrush to contact the base of the defect) abnormality.
- Often involve single tooth with adjacent unaffected tooth.
- Lesions are almost seen on the facial surface and exhibit greater prevalence in those with bruxism.
- Commonly seen in mandibular dentition, presumably because the lingual orientation makes them more susceptible to the concentration of tensile stresses at the cervical region.

Treatment

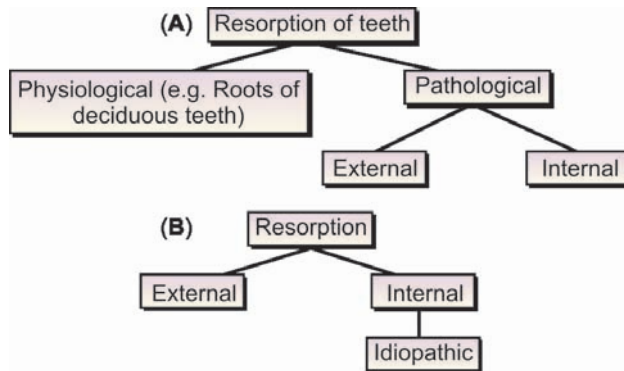
- Restoration should be done on the affected tooth.

RESORPTION OF TEETH

Definition

- It is a chronic progressive damage or loss of tooth structures due to the action of some specialized cells called odontoclasts.

Types of Resorption



PHYSIOLOGICAL RESORPTION

- Occurs in the root portion of deciduous teeth which helps in their natural shedding.

Mechanism

- Chemical substances released by the reduced enamel epithelium (which forms the protective covering for the erupting permanent tooth) causes root resorption of deciduous tooth.
- Deciduous root resorption occurs as an inherent developmental process.
- Pressure exerted by the erupting permanent tooth on the deciduous teeth cause their resorption.

PATHOLOGICAL RESORPTION

- Pathological resorption starts on the root surface or on the crown surface of tooth.

Causes of External Root Resorption

- Periapical Inflammation, often leads to the formation of granulation tissue around the root. It triggers the bone/tooth resorption by stimulating the osteoclast/dentinoclast cells.
- Reimplanted tooth: As the reimplanted or transplanted teeth are always nonvital, these are often resorbed and replaced by bones with subsequent ankylosis.
- Cysts: Pressure exerted by the cysts cause root resorption of the surrounding permanent teeth, (e.g. Dentigerous cyst).
- Tumors: Pressure exerted and chemical mediators released by the tumors cause resorption of roots of the surrounding teeth, (e.g. Ameloblastoma).
- Excessive mechanical or occlusal forces cause trauma from occlusion and excessive orthodontic forces cause injury or necrosis of the periodontal ligaments initiate root resorption.
- Impacted tooth: Pressure exerted by the impacted tooth causes root resorption.

IDIOPATHIC EXTERNAL ROOT RESORPTION (*Burrowing resorption*)

- Commonly seen in relation to a single or multiple erupted teeth.
- Initially a localized area of the root near the cervical region is resorbed.
- The resorption process burrows deeply into and ramifies through out dentin producing a 'labyrinthine network' of lacunae and channels.
- The resorbed tooth structures are replaced by granulation tissue and ankylosis develops.
- The teeth most commonly involved by root resorption were the maxillary bicusps, while the mandibular incisors and molars exhibits the least resorption.

Radiographic Features

- Loss of continuity in lamina dura.
- Appears as carious lesions.
- Raggedness or blunting of the root apex.
- Larger lesions may even produce a '**moth-eaten**' appearance.

Consequences

- Loss of vitality of the affected tooth - endodontic therapy can be effective.
- Fracture of the tooth.

EXTERNAL RESORPTION

Causes

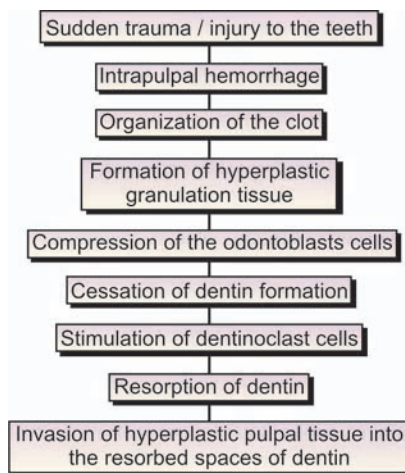
- Periapical inflammation.
- Reimplantation of teeth.
- Excessive mechanical/occlusal forces.
- Tumor and cysts.
- Impaction of teeth.
- Idiopathic.

INTERNAL RESORPTION

(Chronic perforating hyperplasia of the pulp, internal granuloma, odontoclastoma, pink tooth of mummery)

- Pathological resorption of tooth, starting from the pulpal tissue is called internal resorption.

Pathogenesis



TYPES OF INTERNAL RESORPTION

- (a) Internal inflammatory resorption.
- (b) Internal replacement resorption.

(a) Internal Inflammatory Resorption

- Occurs due to an intense inflammatory reaction within the pulp tissue. Microscopically few dentinoclasts are seen.
- Radiographically uniform, spherical enlargement of the root canal are seen.

Internal Replacement Resorption

- Occurs in the absence of any inflammatory reaction within the pulp. Microscopically, fibrous granulation tissue containing ectopic, bone like calcified masses are seen.
- Radiographically, irregular, radiolucency, moth-eaten appearance within dentin.

Clinical Features

- It may involve either crown or root portion of any tooth.
- Initially there is no symptom of the disease. In advanced stages, when the coronal dentin is involved, the tooth appears pink in color (pink spot) as the hyperplastic, highly vascular pulp tissue is seen through the transparent enamel.
- Affected tooth remains vital unless there is no pulp necrosis.
- If left untreated leads to fracture of the teeth.

Radiological Features

- Well defined, spherical shaped, radiolucent area in dentin.
- When perforation occurs, radiograph shows a communication between the pulp chamber and the periodontal ligament space.

Histologic Features

- Single or multiple, irregular areas of resorption in the pulpal surface of dentin.
- Multiple multinucleated giant cells are found in the pulpal tissue.

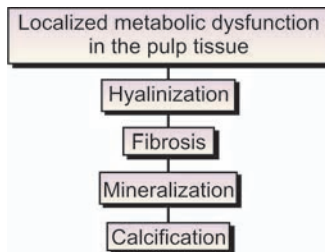
Treatment

- Extirpation of pulp tissue and endodontic therapy.
- When the tooth is perforated, only extraction is possible.

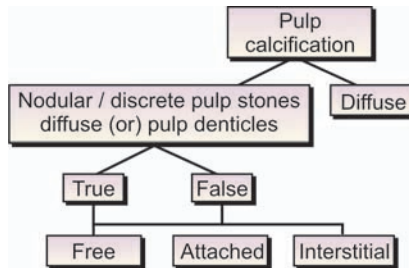
PULP CALCIFICATION

- Deposition of calcified mass within the pulp canal.

Pathogenesis



Types



- **True pulp stones** - Composed of dentinal tubules.
- **False pulp stones** - No tubular structure.
- **Free pulp stones** - Not attached to dentinal wall.
- **Attached pulp stones** - Attached to the dentinal wall of pulp chamber.
- **Interstitial pulp stones** - Pulp stones surrounded by secondary dentin.

Clinical and Radiographic Features

- Denticles or pulp stones can reach sufficient size which can be detected on intraoral radiographs as radiopaque mass with in the pulp chamber or canal.

- Diffuse calcifications are not detectable radiographically. Affected tooth may be vital.
- It may interfere in the endodontic procedure.
- Rarely if the size is very large then it may interfere in the root formation due to early periodontal destruction and tooth loss.

Pulpal Calcification may be Associated with Certain Diseases:

- Dentin dysplasia type II.
- Pulpal dysplasia.
- Tumoral calcinosis.
- Calcinosis universalis.
- Ehlers-Danlos syndrome.

Histologic Features

- Denticles consist of tubular dentin surrounding a central nest of epithelium.
- Later the epithelium degenerates and the tubule undergo sclerosis. Most are attached or embedded.
- Free type occasionally develop outer layers of irregular fibrillar calcification or lamellated type similar to those seen in pulp stones.
- Pulp stones demonstrate a central amorphous mass of irregular calcification surrounded by concentric lamellar rings of regular calcified material.

Diagnosis

- Done by radiographs, ground section preparation and microscopic examination.

HYPERCEMENTOSIS

(Cemental hyperplasia)

Definition

- Increase in thickness of cementum (especially cellular cementum) on the root surfaces of tooth due to excessive cementogenesis.

Etiology

- Periapical inflammation: Stimulates cementoblasts cells and produce localized “Knob - like” overgrowth on the root surface.

- Mechanical stimulation (excessive orthodontic force).
- Functionless/unerupted tooth.
- Paget's disease of bone.
- Tooth repair.
- Gigantism.
- Acromegaly.
- Occlusal trauma.
- Idiopathic.

Clinical Features

- Asymptomatic

Radiographic Features

- Excessive cemental thickness.

Histologic Features

- Excessive disposition of normal cellular or acellular cementum on the root surface.

Clinical Significance

- Concrecence of teeth.
- Ankylosis of tooth.

AGE CHANGES IN ENAMEL, DENTIN AND PULP

(a) Enamel

- More brittle with increasing age
- Less permeable
- Darkened
- Attrition
- Decrease in water content
- Increase in fluoride content
- Decrease in caries incidence.

(b) Dentin

- Increase formation of secondary dentin.
- Dentinal sclerosis.

- Increased translucency of the roots starts with the apex and extends coronally with age (**guideline in forensic odontology to determine age**).

(c) Cementum

- Hypercementosis
- Areas of cemental resorption with increasing age.

(d) Pulp

- Decreased cellularity
- Decreased vascularity
- Increased fibrosis
- Decreased healing potential
- Decreased pulp volume due secondary dentin and pulp calcification.

CEMENTICLES

- Small-calcified bodies lying free in the periodontal ligament formed as a result of dystrophic calcification.

Pathogenesis

- Calcification of epithelial cell rests of Malassez
- Calcification of Sharpey's fibers
- Calcification of thrombosed blood vessels. Cementicles are not visible in radiographs and they do not have clinical significance.

DENTINAL SCLEROSIS

(a) Transparent Dentin

- Calcification of dentinal tubules. It is caused by injury to dentin by caries, ageing process, abrasion or erosion of teeth. Dentinal sclerosis presents as translucent zone in transmitted light. Sclerotic dentin is more highly calcified than normal dentin.

(b) Dead Tracts

- Seen as black zone in transmitted light and white zone by reflected light.

Secondary Dentin

(Irregular dentin/adventitious dentin)

- Formed after deposition of primary dentin. It develops due to increased irritation to pulp.

Clinical Features

- Decreased tooth sensitivity with increased secondary dentin formation. Anterior teeth have higher incidence of secondary dentin formation than molars.

Radiographic Features

- Decrease in size of the pulp chambers and root canals.

Histologic Features

- Fewer dentinal tubules, irregular in nature, tortuous in appearance, formed at a rapid rate. Occasionally, odontoblasts may become entrapped producing a superficial resemblance to bone. Such calcified tissue has been termed as “Osteodentin”.

Bacterial Infections of Oral Cavity

Certain bacteria produce infections in the oral cavity which is manifested in and around oral and paraoral structures. Specific microorganisms produce specific diseases. Some diseases are clinically specific but many number of microorganisms are involved in the pathogenesis. Bacterial infections may be specific to oral cavity or may be manifested systemically.

What are the Bacteria?

Bacteria are prokaryotic microorganisms that do not contain chlorophyll. They are unicellular and do not show true branching except in the so-called higher bacteria.

SCARLET FEVER

(Scarlatina)

- Scarlet fever is a systemic infection produced by group A β -hemolytic streptococci. The disease begins as a streptococcal tonsillitis with pharyngitis in which the organisms elaborate an erythrogenic toxin that attacks the blood vessels and produces the skin rash.

Clinical Features

- Most common in children.
- Incubation period is 3 - 5 days.
- Pharyngitis, tonsillitis, headache, chills, fever, vomiting.
- Enlargement and tenderness of the regional cervical lymphnodes.
- Characteristic diffuse, bright, scarlet skin rash referred as, "Sunburn with goose pimples" appears on the 2nd or 3rd day, particularly in the area of skin folds due to toxic injury to the vascular endothelium produces dilatation of small blood vessels. Hyperemia results known as pastias lines.
- Rash typically begins on upper trunk then spread to extremities sparing the palm and sole.

Oral Manifestations

- Oral mucosa particularly palate may appear congested and the throat is fiery red. The tonsils and faucial pillars are usually swollen and covered with a grayish exudate - "stomatitis scarlatina".
- In the early stage, the tongue exhibits a white coating fine fungiform papillae are edematous, hyperemic, projecting above the surface as small red knobs → "Strawberry tongue".
- By the 4th or 5th day, the coating is lost and the tongue becomes deep red, glistening and smooth with swollen, hyperemic papillae called as "Raspberry tongue".
- After a week, desquamation of the skin occurs, which shows the termination of the disease.

Diagnosis

- Culture of throat secretions.
- Detection of antigens that are specific for group A β -hemolytic streptococci.
- Dick test is done.

Treatment and Prognosis

- Use of antibiotics to treat the disease as well as to aid in preventing the possible complications.
- Mupirocins ointment for topical relief.

Complications

- Peritonsillar abscess
- Rhinitis and sinusitis
- Otitis media and mastoiditis
- Meningitis
- Pneumonia
- Glomerulonephritis
- Rheumatic fever
- Arthritis.

DIPHTHERIA

- Diphtheria is an acute contagious, life threatening infection caused by a gram-positive bacillus, *Corynebacterium diphtheria* or the Klebs Loeffler bacillus.
- Humans are the sole reservoir and the infection is acquired through contact with an infected person (carrier).
- The bacterium produces a lethal exotoxin that causes tissue necrosis, producing nutrients for further growth leading to peripheral spread.
- Mainly spread through droplet.

Clinical Features

- Commonly occurs in children during winter.
- Incubation period is only few days.
- Initial symptoms include restlessness, low-grade fever, headache, malaise, anorexia, sore throat and vomiting .
- Cervical lymphadenopathy associated with an edematous neck enlargement known as “Bull neck”.
- Affects mucosal surfaces and produce exudates of the nasal, tonsillar, pharyngeal, laryngotracheal, conjunctival or genital areas.

Oral Manifestations

- Oropharyngeal exudate begins on the tonsils as a patchy, yellowish-white, thin film which thickens to form an adherent gray covering known as “diphtheritic membrane”.

- The superficial epithelium is an integral part of this exudate and leaves a bleeding surface if stripped away.
- The diphtheritic membrane may involve the soft palate, uvula, larynx and trachea resulting in stridor and respiratory difficulties → “Diphtheritic cough”.
- The soft palate paralysis can lead to nasal regurgitation during swallowing.

Diagnosis

- Culture specimen should be collected from the surface or underneath the diphtheritic membrane and also from the nose.

Treatment and Prognosis

- Can be prevented by prophylactic active immunization.
- Antitoxin in combination with antibiotics to prevent further toxin production to stop the local infection and to prevent transmission.

Complications

- Myocarditis
- Polyneuritis
- Acute interstitial nephritis.

TUBERCULOSIS

- Tuberculosis is an infectious granulomatous disease caused by the acid-fast bacillus *Mycobacterium tuberculosis* or rarely *Mycobacterium bovis*.
- Most infections are the result of direct person-to-person spread through airborne droplets from a patient with active disease. Primary tuberculosis occurs in previously unexposed people and involves the lung which elicits a nonspecific chronic inflammatory reaction. Active disease develops later in life from the reactivation of organisms in a previously infected person and associated with compromised host defence → Secondary tuberculosis. Diffuse dissemination through vascular system is miliary tuberculosis.

Clinical Features

- Episodic fever and chills.
- Easy fatigability and malaise.
- Gradual loss of weight, night sweats.
- Persistent cough with or without associated hemoptysis.
- Tuberculous infection of submaxillary and cervical lymph nodes known as scrofula, may progress to form an actual abscess which perforate and discharge pus.
- Areas of nodal involvement, may present as calcified lymph nodes radiographically.
- *Lupus vulgaris*, which is primary tuberculosis of the skin appears as papular nodules which frequently ulcerate.

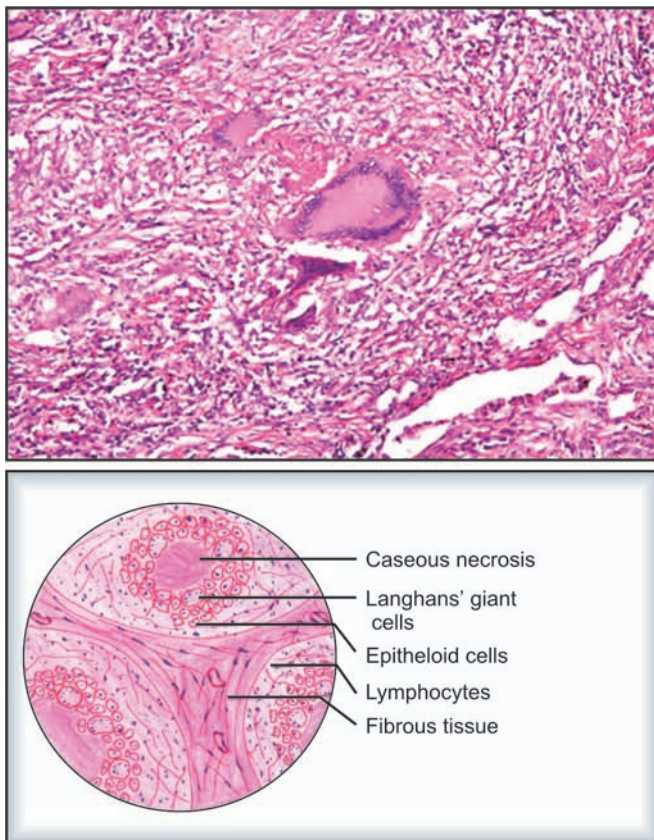


Fig. 7.1: Tuberculosis

Oral Manifestations

- Oral lesions are uncommon but can occur as nodular, granular ulcerated or firm leukoplakic areas.
- Most of the lesions are secondary infection from primary lesion either through hematogenous spread or from exposure to infected sputum.
- Tongue is commonly affected.
- Oral lesions appear as an irregular, superficial or deep painful ulcer.
- *Tuberculous gingivitis* appears as a diffuse, hyperemic, nodular or papillary proliferation of the gingival tissues.
- Tuberculous periapical granuloma or tuberculoma occurs when the microorganisms enter the periapical tissues by direct immigration through pulp chamber of a tooth with an open cavity.
- Tuberculous osteomyelitis involving the jaws occurs in the later stages of the disease.

Histologic Features

- Areas of infection demonstrate the formation of granuloma (tubercle) which are circumscribed collections of epithelioid histocytes, lymphocytes and multinucleated giant cell with central caseous necrosis.

Diagnosis

- Tuberculin skin test (Mantoux test) which shows a cell-mediated hypersensitivity reaction to tuberculin antigen.
- Confirmed by special mycobacterial stains (Ziehl - Neelsen stain) or culture of infected specimen or tissue.

Treatment and Prognosis

- Multi-agent therapy is the treatment of choice
- Treatment of oral tuberculosis is secondary to treatment of the primary lesions.

LEPROSY

(Hansen's disease)

- Leprosy is a chronic granulomatous infection caused by an acid fast bacillus, *Mycobacterium leprae*. The organism has low infectivity and exposure rarely results in clinical disease.

Classification

Related to the immune reaction to the organism. Two main clinical forms occur.

- **Tuberculoid Leprosy**
 - Occurs in patients with a high immune reaction.
 - Organisms are not found in skin biopsy specimens.
 - Lepromin test is positive.
 - Usually localized.
- **Lepromatous leprosy**
 - Seen in patients with an absence of cell mediated immune response.
 - Exhibit numerous organisms in tissue.
 - Negative lepromin test.
 - Exhibit diffuse disease.

Clinical Features

- **Tuberculoid leprosy**
 - Incubation period is 2-5 years.
 - Presents with a small number of well circumscribed, hypopigmented skin lesions.
 - Single or multiple macular, erythematous eruptions, with dermal nerve and peripheral nerve trunk involvement resulting in loss of sensation.
- **Lepromatous leprosy**
 - Incubation period is 8-12 years. Begins slowly with numerous, ill-defined hypopigmented macules or papules that subsequently lead to progressive thickening of the skin and the characteristic nodules.
 - Produce severe disfigurement (leonine facies).
 - Hair (eyebrows and lashes) is lost.
 - Nerve involvement leads to a loss of sweating and decreased light, touch, pain temperature sensors.
 - Nasal involvement leads to nosebleeding, stuffiness, loss of sense of smell.
 - Collapse of the bridge of nose is pathognomonic.

Oral Manifestations

- Oral lesions are rare in the tuberculoid variant.
- Sites that are cooled by the passage of air are commonly affected.

- Anterior maxillary area, hard and soft palate, uvula, tongue are commonly affected.
- Affected soft tissue presents yellowish to red, sessile, firm, enlarging papules leading to ulceration and necrosis, followed by attempted healing by secondary intention. Continuous infection leads to scarring, loss of tissue.
- Complete loss of the uvula and fixation of the soft palate may occur.
- Lingual lesions appear in the anterior third and begins as areas of erosion which develop into large nodules.
- Infection of the lip results in macrocheilitis.
- Involvement of the anterior maxilla result in bone erosion with loss of teeth in this area - "Facies leprosa".
- In children, it affects developing teeth leading to enamel hypoplasia and short tapering roots.
- Pulp infection leads to internal resorption or pulpal necrosis.
- Teeth with pulpal involvement may show a red discoloration of the crown, due to the intrapulpal vascular damage secondary to the infection.
- Granulomatous involvement of nasal cavity can erode the palatal tissues result in perforation.
- Facial and trigeminal nerve are involved.

Histologic Features

Tuberculoid Leprosy

- Demonstrate well formed granulomatous inflammation, with clusters of epithelioid histiocytes, lymphocytes and multinucleated giant cells.
- Paucity of organisms.

Lepromatous Leprosy

- No well-formed granuloma.
- Typical finding is sheets of lymphocytes intermixed with vacuolated histiocytes known as "*lepra cells*".
- Abundance of organisms.

Treatment and Prognosis

- Specific long-term chemotherapy.
- After resolution of the infection, the therapy must be directed toward reconstruction of the damage in addition to physiotherapy and education.

ACTINOMYCOSIS

- Actinomycosis is a chronic, granulomatous, suppurative and fibrosing disease caused by anaerobic, gram-positive, non acid-fast, branched, filamentous bacteria, the most common being *Actinomyces israeli*.
- Other causative organisms include *Actinomyces naeslundii*, *Actinomyces viscosus*, *Actinomyces odontolyticus* and *Actinomyces propionica*.
- Sites of colonization in healthy patients include the tonsillar crypts, dental plaque and calculus, carious dentin, gingival sulci and periodontal pockets.

Clinical Features

- May present as either an acute, rapidly progressing infection or as a chronic slowly spreading lesion associated with fibrosis.
- Common sites of involvement are the cervicofacial, abdominal, thoracic, cutaneous and the genital regions.
- The disease is characterized chiefly by the formation of abscesses which tend to drain by the formation of sinus tracts. Pus may show the typical “**sulfur granules**” or colonies of organisms that appear as tiny yellow grains in the suppurative material.
- In the cervicofacial region, the organisms enter the tissues through the oral mucous membrane and may either remain localized in the adjacent soft tissues or spread to involve the salivary glands, bone or the skin of the face and neck.
- The classic description is of a “wooden” indurated area of fibrosis, which later forms a central softer area of abscess. The infection may extend to the surface forming a fistula.
- Common sites of involvement are the soft tissues of the submandibular, submental and cheek areas. Most frequently affected site is the area overlying the angle of the mandible.
- Intraductal colonization by the organism may lead to infections

in both the submandibular and parotid gland with abscess formation in the submandibular and masseter spaces.

- Actinomycetic osteomyelitis of the mandible and maxilla can occur. If the maxilla is involved, it may eventually involve the cranium, meninges or the brain.
- Intrabony colonization of dentigerous cysts also occurs.
- Periapical inflammatory lesions involved by bacteria may result in lesions which are difficult to resolve with endodontic treatment.

Histologic Features

- Demonstrates chronically inflamed granulation tissue surrounding large collections of PMN leukocytes and characteristic colonies of microorganisms which appear to be

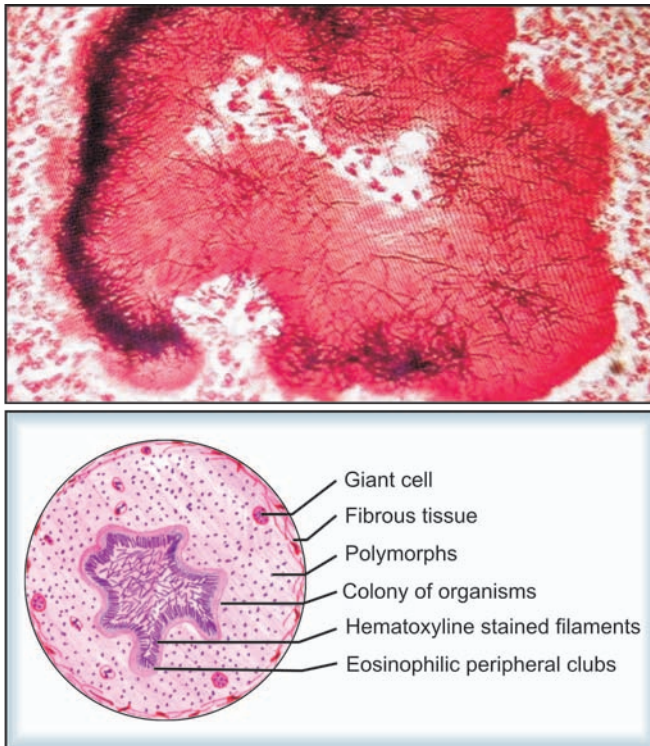


Fig. 7.2: Actinomycosis

floating in a sea of PMN leukocytes, multinucleated giant cells and macrophages.

- The colonies consist of club-shaped filaments that form a radiating rosette pattern.
- With hematoxylin eosin stains, the central core stains basophilic and the peripheral portion eosinophilic, this peculiar appearance of the colonies leads to the term “**ray - fungus**”.

Diagnosis

- Culture test
- Biopsy
- Fluorescein - conjugated antiserum can be used on the granules.

Treatment and Prognosis

- Chronic cases should be treated with a prolonged high dose of antibiotics in association with abscess drainage and excision of the sinus tracts.

Differential Diagnosis

- Botryomycosis, which also shows sulfur granules.

TETANUS

(“*Lock -jaw*”)

- Tetanus is a disease of the nervous system characterized by intense activity of the motor neurons resulting in severe muscle spasm.
- Caused by the exotoxin of the anaerobic gram-positive *Bacillus Clostridium tetani* which acts at the synapse of the interneurons of inhibitory pathways and motor neurons to produce blockade of spinal inhibition.
- The organism enters the body through injury sites.

Clinical Features

- Incubation period varies.
- Pain and stiffness in the jaws and neck muscles, with muscle rigidity producing trismus and dysphagia.

- “Risus sardonicus”—Rigidity of facial muscles.
- Opisthotonus—Rigidity of the entire body.
- Reflex spasms occurs after stimuli.
- “Cephalic tetanus”—Localized or generalized tetanus which occurs in association with cranial nerve palsy, commonly the seventh cranial nerve.
- Acute trismus occurring in tetanus may simulate acute oral infection, trauma and TMJ dysfunction.

Treatment

- Antimicrobial drugs.
- Active and passive immunization.
- Surgical wound care.
- Anticonvulsants if needed.

SYPHILIS

(Lues)

- Syphilis is a chronic infection caused of a spirochete, *Treponema pallidum*.

Classification

(a) Acquired

- Primary
- Secondary
- Tertiary

(b) Congenital

Clinical Features

(a) Acquired Syphilis

- The acquired form is contracted primarily as a venereal disease. Also, persons like dentist working on infected patients in a contagious stage may also acquire the disease.

(i) Primary syphilis

- Becomes clinically evident 2-3 weeks after initial inoculation.
- During this stage, the organism is spreading systemically through the lymphatic channels.

- Characterized by the chancre, which is an elevated ulcerated nodule showing local induration and producing regional lymphadenitis.
- The external genitalia and anus are the common sites for a primary lesion.
- Oral lesions are most commonly seen on the lip, tongue, palate, gingival and tonsils.
- The oral lesion presents as a painless, clean-based ulceration or as a vascular proliferation resembling pyogenic granuloma.
- The lesion on the lip may have a brownish crusted appearance.
- If left untreated, the initial lesion heals within 3-8 weeks.

(ii) Secondary syphilis

Metastatic stage

- Arise 6 weeks after the primary lesion and is characterized by painless diffuse macular papular mucocutaneous rash of the skin and mucous membrane including the palmar plantar areas.
- Systemic symptoms like sore throat, malaise, headache, weight loss, fever and musculoskeletal pain.
- The oral lesion, "mucous patches" are usually multiple painless, grayish-white plaques overlying an ulcerated surface and occur on the tongue, gingiva or buccal mucosa. They are ovoid or irregular in shape surrounded by an erythematous zone. Mucous patches are highly infectious.
- Papillary lesions that resemble viral papillomas may arise during the secondary stage and are known as condylomata.
- In the presence of a compromised immune system, secondary syphilis exhibits an explosive and spread form known as lues maligna. Prodromal symptoms include fever, headache, myalgia, followed by formation of necrotic ulcerations involving the face and scalp. Oral lesions are also present. Common in patients with AIDS.
- Spontaneous resolution occurs within 3-12 weeks but relapse may occur after a year.

(iii) Tertiary syphilis

Late syphilis

- Do not usually appear for several years.
- Involves chiefly the cardiovascular system and the central nervous system and is non-infectious.

- CVS
 - Aneurysm of the ascending aorta.
 - Left ventricular hypertrophy.
 - Congestive heart failure.
- CNS
 - Tabes dorsalis
 - Psychosis
 - Dementia
 - Paresis
 - Death.
- Characteristic of the tertiary stage are scattered foci of granulomatous inflammation which may affect the skin, mucosa, soft tissue, bones and internal organs known as “**gumma**”.
- It presents as an indurated, nodular or ulcerated lesion with central necrosis.
- Intraoral involvement mostly affects the tongue and palate.
- Tongue may be diffusely involved and appears large, lobulated and irregularly shaped. Diffuse atrophy and loss of dorsal tongue papillae leads to a condition known as “**luetic glossitis**”.
- Involvement of the palate leads to ulceration which perforates through the nasal cavity.

(b) Congenital Syphilis

- Pregnant woman transmit the infection even in the latent stages. Maternal transmission during the first two stages of infection resulting in miscarriage, still birth, or congenital malformations. Infection of the fetus may occur at any time during pregnancy, but the stigmata begins only after the 4th month of gestation. The clinical changes secondary to fetal infection are known as congenital syphilis.
- The three pathognomonic diagnostic features, as described by sir Jonathan Hutchinson in 1858 is known as “Hutchinson’s triad” with includes.
 - Hutchinson’s teeth → Hutchinson
incisors (screw - driver shaped)
→ Mulberry molars.
 - Interstitial keratitis -opacified corneal surface with resultant loss of vision.
 - Eighth nerve deafness.

The other alterations seen in congenital syphilis include:

- Frontal bossing.
- High - arched palate.
- Saddle nose.
- Mandibular protruberance.
- Higoumenaki's sign (enlargement of clavicle adjacent to the sternum).
- Rhagades (Premature perioral fissuring).
- Saber shin (Anterior bowing of tibia as a result of periostitis).
- Scaphoid scapulae (Concavity of vertebral border of scapulae).
- Clutton's joint (Painless synovitis enlargement of joints, especially the knee).

Histologic Features

- The surface epithelium is ulcerated in primary lesion and may be ulcerated or hyperplastic in secondary stage.
- Increase in the vascular channels in lamina propria.
- Intense chronic, inflammation is present, with predominantly lymphocytes and plasma cells in a perivascular pattern.
- With the use of special silver - impregnation technique, scattered "corkscrew - like" spirochetal organisms are demonstrated.
- Oral tertiary lesions typically exhibit surface ulcerations with peripheral pseudoepitheliomatous hyperplasia. The underlying inflammatory infiltrate usually demonstrate foci of granulomatous inflammation with well circumscribed collection of histiocytes and multinucleated giant cells.
- In the tertiary stage, even with the use of special stain, the organisms can not be demonstrated.

Diagnosis

- Dark-field examination of a smear.
- Specific immunofluorescent antibody test or serologic test.
- VDRL and RPR (Rapid plasma reagin) test.
- FTA - ABS and TPHA tests.

Treatment and Prognosis

- Antibiotic therapy.

GONORRHEA

- Gonorrhea is primarily a venereal disease produced by *Neisseria gonorrhoeae*, a gram-negative bacteria.

Oral Manifestations

- Extragenital gonorrheal infection of the oral cavity occurs as a result of oral-genital contact or inoculation through infected hands.
- The lips may develop acute painful ulcerations limiting motion.
- The gingiva may become erythematous, with or without necrosis.
- The tongue may show red, dry ulcerations or become glazed swollen with painful erosions.
- Similar lesions occur on buccal mucosa and palate.
- Gonococcal pharyngitis and tonsillitis also occur which appear as vesicles or ulcers with a gray or white pseudomembrane.
- Gonococcal parotitis occurs as a result of an ascending infection from the duct to the glands.

Diagnosis

- Gram staining.
- Culture and sugar fermentation test.
- Fluorescent antibody test.

Treatment

- Antibiotic therapy.

GRANULOMA INGUINALE

(*Granuloma venereum, donovanosis*)

- Granuloma inguinale is a chronic, infectious granulomatous disease caused by microorganisms probably bacilli, formerly known as *Donovania granulomatis* and recently as *Calymmatobacterium granulomatis*.

Clinical Features

- The primary lesion appear on the external genitalia and inguinal region and manifest as papules or nodules which ulcerate to form clean, granular lesions with rolled margins with a tendency for peripheral enlargement.
- Satellite lesions arise through lymphatic extension.
- Inguinal ulceration arises as a fluctuant swelling known as pseudo bulb.

Oral Manifestations

- Oral lesions may appear on the lips, buccal mucosa or palate.
- Classified into three types based on varied clinical appearances.

Histologic Features

- Granulation tissue with infiltration of PMN leukocytes and plasma cells.
- Pseudo epitheliomatous hyperplasia.
- Presence of large mononuclear phagocytes containing tiny intracytoplasmic cysts.
- Donovan bodies are seen, characterized by tiny, elongated basophilic and argyrophilic rods present in profuse numbers within the macrophages.

Treatment and Prognosis

- Antibiotic therapy, resolves genital lesion along with oral lesion.
- Exacerbation of genital lesions results in worsening of oral lesions.

MIDLINE LETHAL GRANULOMA

(Malignant granuloma; lethal granuloma)

- The lethal granuloma is a serious but uncommon infection, described as an idiopathic, progressive destruction of the nose, paranasal sinuses, palate face and pharynx.
- The affected person characteristically appears to exhibit complete lack of resistance to the insidious progress of the disease.

Etiology

- Lethal granuloma is due to a dysfunction of the immune mechanism normally responsible for granuloma formation . A vascular allergy occurs, either the arthus phenomenon or periarteritis nodosa, depending on whether capillaries or arterioles are affected and hyperimmune tissue become necrotic because of obstruction of the blood supply represents a fulminant hypersensitivity response to an unidentified antigen.
- Diseases which may appear clinically as Midline Lethal Granuloma are:

Bacterial

- Brucellosis
- Rhinoscleroma
- Leprosy
- Actinomycosis
- Tuberculosis
- Syphilis
- Short maxilla.

Fungal

- Histoplasmosis
- Candidiasis
- Coccidiomycosis
- Blastomycosis
- Rhinosporidiosis
- Phycomycosis.

Parasitic

- Leishmaniasis
- Myiasis.

Neoplastic Diseases

- Squamous cell carcinoma
- Rhabdomyosarcoma
- Polymorphic reticulosis/lymphomatoid granulomatosis
- Conventional lymphoma.

Clinical Features

- Lesion begins as superficial ulceration of the palate or nasal septum with a feeling of stuffiness in the nose.
- Ulceration spreads from the palate to the inside of the nose and then to outside.
- The palatal, nasal and malar bones may be involved which undergo necrosis and then sequestrate.
- Purulent discharge from the eyes and nose.
- Formation of perforating sinus tracts.
- Soft tissue of the slough away, leaving a direct opening into nasopharynx and oral cavity.
- Death may occur due to exhaustion or of hemorrhage due to involvement of large blood vessel.
- The overlying skin becomes inflamed, edematous and necrotic forming a line.

Histologic Features

- Affected tissue reveals extensive necrosis with infiltration of inflammatory cells and formation of new capillaries.

Treatment

- Antibiotics along with corticosteroid.
- Radiation therapy.

Inflammatory Diseases of Unknown Etiology

- Wegener's granulomatosis.
- Idiopathic granulomatosis.
- Idiopathic midline destructive disease.

WEGENER'S GRANULOMATOSIS

- Wegener's granulomatosis is a disease of unknown etiology, although an aberrant hypersensitivity reaction to an unknown antigen has been told as a cause.

Clinical Features

- Is a multi system disease mainly involving the vascular, renal and respiratory system.

- Initially, rhinitis, sinusitis and otitis or ocular symptoms develop.
- Later, cough, hemoptysis, fever, joint pain and hemorrhagic or vesicular skin lesions develop.
- Chest X-ray reveals granulomatous lesions of the lungs.
- Glomerulonephritis develops which leads to uremic terminal renal failure or vesicular skin lesions develop.
- Chest X-ray reveals granulomatous lesions of the lungs.
- Glomerulonephritis develops with leads to uremia and terminal renal failure.

Oral Manifestations

- Gingival lesions may be ulcerations, friable granular lesions or gingival enlargements.
- Ulceration of the palate through extension from nose with destruction of the nasal septum.
- Small ulcerations resembling aphthae.
- Diffuse ulcerative stomatitis is also seen.
- Spontaneous exfoliation of teeth.
- Failure of healing of extraction sockets.

Lab Findings

- Anemia, leukocytosis, elevated ESR, hyperglobulinemia, hematuria.

Histologic Features

- Upper respiratory tract and lung lesions consist of giant cell necrotizing granulomatous lesions which show vasculitis.
- Other lesions show a similar, nonspecific granulomatous process with scattered giant cells.

Treatment

- Use of cytotoxic agents.

NOMA

(Cancrum oris; gangrenous stomatitis)

- Noma is a rapidly progressive, opportunistic infection caused by components of the normal oral flora, which become pathogenic during periods of compromised immune status.

- *Fusobacterium nucleatum* forms a symbiotic relationship with one or more bacterial organisms, of which the most commonly implicated are *Borrelia vincentii*, *Staphylococcus aureus*, *Prevotella intermedia* and nonhemolytic streptococci.

Predisposing Factors

- Malnutrition
- Dehydration
- Poor oral hygiene
- Malignancy
- An immunodeficiency disorder, like AIDS.
- Recent illness—diphtheria, measles, pneumonia, scarlet fever, syphilis, tuberculosis, blood disorders.
- Commonly, the infection begins as acute necrotizing ulcerative gingivitis.

Clinical Features

- Arise in children aged 2 - 10 years.
- Begins on the gingiva as ANUG and extends to involve the adjacent soft tissue to form acute necrotizing ulcerative mucositis.
- Zones of necrosis may develop in soft tissues not continuous with the gingiva in areas of trauma.
- The initial site is commonly an area of stagnation around a fixed bridge or crown. Demarcation between healthy and large masses of the tissue may slough out, exposing the jawbone. Osteomyelitis may occur.
- The formation of gangrene is denoted by the appearance of blackening of the skin.
- Fetid odour, significant pain, fever, malaise with regional lymphadenopathy are typical.
- Patient may suffer secondary infection and may die from toxemia or pneumonia.

Treatment and Prognosis

- Appropriate antibiotics along with therapeutic attention and adequate nutrition, hydration and electrolyte in balances.
- Reconstruction should be delayed for one year to ensure complete recovery.

- Facial disfigurement with effects on future of growth development occurs.
- Trismus from significant scarring associated with mandibular involvement can occur.

PYOGENIC GRANULOMA

(Granuloma pyogenicum)

- *Pyogenic granuloma* occurs as a response of the tissues to a nonspecific infection.

Etiology

- Botryomycotic infection—infection of horses transmitted to man.
- Streptococcal or staphylococcal infection.
- Generally, accepted concept is that, it is a result of minor trauma to the tissue which provides a pathway for the invasion of nonspecific types of microorganisms.

Clinical Features

- In the oral cavity, arises most commonly in the gingiva followed by the lips, tongue and buccal mucosa.
- The lesion is an elevated, pedunculated or sessile mass with a smooth, lobulated or even a warty surface which is ulcerated and shows a tendency for hemorrhage either spontaneously or upon slight trauma.
- The lesion is deep red or reddish purple and painless soft in consistency.
- Sizes may vary from a few mm to cm.

Histologic Features

- Similar to granulation tissue except that it is exuberant and well localized.
- Overlying epithelium is thin and atrophic or hyperplastic.
- A fibrinous exudate may be present if the lesion is ulcerated.
- Occurrence of vast numbers of endothelium - lined vascular spaces and extreme proliferation of fibroblast and budding endothelial cells.
- Infiltration of PMN leukocytes, lymphocytes and plasma cells.

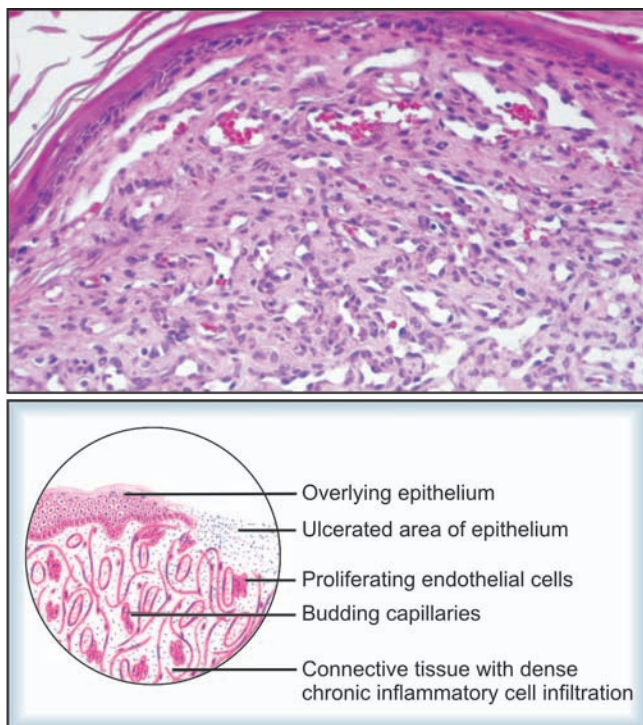


Fig. 7.3: Pyogenic granuloma

- Gradual obliteration of capillaries may occur if the lesion is not surgically excised.
- An old lesion may resemble a fibroepithelial polyp or a typical fibroma.

Pregnancy Tumor

- It is a well defined lesion which appears about the 3rd month of pregnancy and gradually increases in size.
- May or may not regress after delivery.
- During pregnancy if surgically removed, may recur.
- Pregnancy tumour is a type of pyogenic granuloma which occurs as a result of local minor trauma or irritation in which the tissue reaction is intensified by the endocrine alteration occurring during pregnancy.

Treatment and Prognosis

- Surgical excision.
- When excising a *Pyogenic granuloma* of the gingiva extreme care should be taken to scale the adjacent tooth, since the calculus may act as the irritation leading to recurrence.

PYOSTOMATITIS VEGETANS

- *Pyostomatitis vegetans* is an uncommon inflammatory disease of the oral cavity and is one part of a syndrome in which the patients also manifest ulcerative colitis or other gastrointestinal disturbances.
- Regional enteritis or Crohn's disease may also be associated with *Pyostomatitis vegetans*.

Oral Manifestations

- The oral lesions consists of large numbers of broad based papillary projections, tiny abscesses or vegetations developing in areas of intense erythema.
- Lesions may be red or pink in color which on careful examination show tiny pustules beneath the epithelium which discharge purulent material on rupturing and leave areas of ulceration which may coalesce into larger areas of necrosis.
- Buccal mucosa is commonly affected and shows a "Cobblestone" appearance vestibular lesions appear as folds and ulcers, lips appear swollen and indurated. Gingiva and alveolar mucosal lesions appear granular and erythematous. Palatal lesions show multiple aphthous ulcers.

Histologic Features

- Papillary projections show an intact stratified squamous epithelium with underlying loose connective tissue infiltrated with plasma cells, lymphocytes. PMN leukocytes, eosinophils.
- Tiny areas of focal necrosis and microabscess formation is often seen.

Treatment

- Oral lesions are refractory to antibiotics.
- Oral lesions resolve when the intestinal disturbance is controlled.

Viral Infections of Oral Cavity

Virus is a latin word which means venom or poison. These are submicroscopic elements which is seen in specific living cells and can be introduced into these host cells. Viruses may affect anything in this world. Size of the viruses varies in millimicrons. Virus consists of a central core of DNA or RNA bat never both, which is surrounded by a caspid made up of proteins or enmassed by a glycoproteins and lipids derived from host cell membranes.

Virally infected cells produce the same type of nucleic acid, so the susceptibility of cells infection may be increased. viruses cause certain infectious diseases and produce long - lasting immunity against reinfection by the same viruses.

Viruses are also Involved on Neoplastic Conditions.

Classification of viruses are based upon the biologic, chemical, physical properties, type of nucleic acid, size, shape and substructure of the particle by International committee on nomendature of viruses of the international association of microbiological societies. Diagnosis of virus by lab investigation us loco and difficult, so diagnosed by their clinical presentation.

Viruses have been defined as submicroscopic entities which reproduce only within specific living cells and which can be introduced into these host cells.

CLASSIFICATION OF MAJOR VIRUS GROUPS AND VIRUS DISEASES

- *RNA viruses*
- *Orthomyxovirus*
 - Influenza
- *Paramyxovirus*
 - Measles (rubeola)
 - Mumps.
- *Rhabdovirus*
 - Rabies
 - Hemorrhagic fever.
- *Arenavirus*
 - Lymphocytic choriomeningitis
 - Lassa fever.
- *Calicivirus*
- *Coronavirus*
 - Upper respiratory infection.
- *Gunyavirus*
 - Poliomyelitis
 - Coxsackie disease
 - Common cold
 - Foot and mouth disease
 - Encephalomyocarditis.
- *Reovirus*
- *Togavirus*
 - Rubella
 - Yellow fever
 - St. Louis encephalitis
 - K. retrovirus (RNA tumor virus).
- *DNA Virus*
 - Herpesvirus
 - a. Herpes simplex
 - b. Varicella/herpes zoster
 - c. Cytomegalic inclusion disease
 - d. Epstein-Barr virus—Infectious mononucleosis.
 - Poxvirus
 - a. Small pox
 - b. *Molluscum contagiosum*.

- Adenovirus
 - a. Pharyngoconjunctival fever.
 - b. Epidermic keratoconjunctivitis.
- Parvovirus
- Iridovirus
- Papovavirus
 - a. Human warts or papillomas
 - b. Tumorigenic viruses in animals.

HERPES SIMPLEX

(Acute herpetic gingivostomatitis; herpes labialis; fever blisters; cold sores)

- Herpes simplex is an acute infectious disease and is the most common viral disease affecting man.
- The tissues involved are derived from the ectoderm and consist principally of the skin, mucous membranes, eyes and central nervous system.
- There are two immunologically different types of HSV.
 - HSV 1 → spreads predominantly through infected saliva or active perioral lesions. Most commonly involved are the intraoral sites, lips, eyes and skin above the waist.
 - HSV 2 → transmitted predominantly through sexual contact and involves the genitalia and skin below the waist.
- Other viruses include.
 - Varicella - zoster virus (VZV).
 - Epstein - Barr virus (EBV).
 - Cytomegalovirus (CMV).
- Herpes genitalis, caused by HSV 2 is a common disease of the uterine cervix, vagina, vulva and penis. The type 2 virus of genital herpes is more virulent than type 1 and is associated with carcinoma of the uterine cervix. Because of changing sexual practices in recent years, there is widespread translocation in the usual habitats of type 1 and type 2 HSV.
- Herpetic meningeal encephalitis, is a serious form of infection, characterized by sudden fever and symptoms of increased intracranial pressure. Paralysis of muscles occurs, while convulsions and death may ensue.
- Herpetic conjunctivitis or keratoconjunctivitis is a common disease characterized by swelling and congestion of the palpebral

conjunctiva. Keratitis and corneal ulceration may also occur. Herpetic vesicles of the eyelids are typical, but these lesions heal rapidly.

- Herpetic eczema (Kaposi's varicelliform eruption) is an epidermal form of the herpetic infection superimposed upon a pre-existing eczema. Characterized by diffuse vesicular lesions of the skin. This herpetic infection may also be superimposed on severe seborrheic dermatitis, impetigo, scabies, Darier's disease and pemphigus. Usually children are affected, exhibit a high fever with typical umbilicated vesicles and other systemic manifestations and is fatal.
- Disseminated herpes simplex of the newborn is a disease in which the infant acquires the infection during passage through the birth canal of the mother with herpetic vulvovaginitis. Infants manifest the disease by the 4th-7th day of life and exhibit various signs and symptoms of the disease, some may die on 9th-12th day or if it survive may show residual neurologic involvement.

PRIMARY HERPETIC STOMATITIS

Clinical Features

- The initial exposure of an individual without antibodies to the virus is called primary infection.
- Rarely occurs before the age of 6 months, because of the presence of circulating antibodies in the infant derived from the mother.
- Abrupt onset accompanied by anterior cervical lymphadenopathy, fever, anorexia, irritability, pain upon swallowing.
- Sore mouth lesion develop and involve the lips, tongue, buccal mucosa, palate, pharynx and tonsils. Initially, the affected mucosa develops numerous pinhead vesicles, which rapidly collapse to form, numerous small red lesions. These lesion enlarge slightly and develop central mass of ulceration, covered by yellow fibrin. The gingiva is enlarged painful and extremely erythematous and also exhibits distinctive, punched-out erosions along the mid-facial free gingival margin.
- Satellite vesicles of the perioral skin is common.
- These lesions heal spontaneously within 7 - 14 days without any scar formation.

- HSV does not remain latent at the site of original infection. The virus is taken up by the sensory nerves and transported to the associated sensory or autonomic ganglia. With oral HSV - 1 infection, the trigeminal ganglion is colonized and in case of HSV - 2 lumbosacral ganglion is involved and the virus remains at this site in a latent stage. The virus uses the axons of the sensory neurons to travel back and forth to the peripheral skin or mucosa.

Histologic Features

- The herpetic vesicle is an intra epithelial blister filled with fluid.
- The degenerating epithelial cells exhibit acantholysis, nuclear clearing and nuclear enlargement known as “ballooning degeneration”, while others characteristically contain intra-nuclear inclusions known as lipschutz bodies. These are eosinophilic, ovoid, homogenous structures within the nucleus which tend to displace the nucleus and nuclear chromatin peripherally, which produces a peri-inclusion halo.
- Connective tissue infiltrated with inflammatory cells.
- The lesions heal by peripheral epithelial proliferation.

Modes of Transmission

- Droplet infection
- Direct contact
- Associated with pneumonia, meningitis and common cold.

Treatment

- Treatment is only supportive and symptomatic antibiotic therapy aids in the prevention of secondary infection.

RECURRENT OR SECONDARY, HERPETIC LABIALIS AND STOMATITIS

- Recurrent herpetic stomatitis occurring in adults manifests clinically as an attenuated form of the primary disease.
- Compared with the lower socioeconomic level, those in the higher socioeconomic level, especially medical, dental and nursing personnel are at higher risk of contact and infection due to lack of circulating HSV antibodies.

Reactivation of virus occurs due to a variety of conditions which include.

- Old age
- Ultraviolet light
- Emotional stress
- Pregnancy
- Allergy
- Trauma
- Respiratory illnesses
- Menstruation
- Systemic diseases
- Malignancy
- Gastrointestinal disturbances.

Mode of Action

- When reactivation is triggered, the viruses which reside dormant within the regional ganglia spread along the nerves to sites on the oral mucosa and skin where they destroy the epithelial cells and induce the typical inflammatory response with the characteristic lesions of recurrent infection.

Clinical Features

- Recurrent infections may occur either at the site of primary inoculation or in adjacent areas of surface epithelium supplied by the involved ganglion.
- May occur at widely varying intervals.
- Most common site of recurrence is the vermilion border and adjacent skin of the lips and is known as herpes labialis (“cold sore” or “fever blister”).
- Prodromal signs and symptoms include pain, burning, itching, tingling, a localized warmth, swelling or slight soreness of the location.
- Multiple small, erythematous papules develop and form clusters of fluid vesicles, which rupture quickly leaving a small red ulceration with a slight erythematous halo.
- On the lips the ruptured vesicles become covered by a brownish crust.

- Involvement of the oral mucosa is limited to the keratinized mucosa (attached gingiva and hard palate) small vesicles form, which rapidly collapse to form a cluster of erythematous macules, which may coalesce or slightly enlarge. The damaged epithelium is lost and a central yellowish area of ulceration develops.
- The lesions gradually heal within 7 -10 days and leave no scar.
- Infection of the thumb or fingers is known as herpetic whitlow (herpetic paronychia) which may occur as a result of self -inoculation in children with orofacial herpes or in medical or dental personnel due to infection from contact with infected patients.
- Recurrences on the digits may result in paresthesia and permanent scarring.

Histologic Features

- Ballooning degeneration, chromatin margination and lipschutz bodies as seen in primary infection along with the characteristic multinucleated giant cells or Tzanck cells.

Laboratory Findings

- Diagnostic methods for viral herpes include.
 - Viral isolation and identification in various system.
 - Immunofluorescent staining of smears, impressions or cryostat sections with fluorescein-labelled HSV protein or antibody protein.
 - Immunoperoxidase technique.
 - Serologic assays.

Treatment

- Specific antiviral chemotherapeutic agents are available which include.
 - Acyclovir
 - Vidarabine
 - Idoxuridine.
- Intraoral lesions can be treated with chlorhexidine (alone or in combination with acyclovir suspension).
- Resistant strains are treated with trisodium phosphonoformate hexahydrate (foscarnet), but has significant side-effects.

Differential Diagnosis

- Herpes zoster impetigo, erythema multiforme, small pox, pemphigus, epidermolysis bullosa, food or drug allergy, etc.

RECURRENT APHTHOUS STOMATITIS

(*Aphthous ulcers; aphthae; canker sores*)

- Recurrent aphthous stomatitis is a common disease characterized by the development of painful, recurring solitary or multiple ulceration of the oral mucosa.
- Because of the similarity between this disease and herpes simplex infection based on precipitating factors, clinical appearance, duration of lesions, chronic recurrence and general failure of response to any form of therapy, there is a confusion between these two diseases.
- There is no etiologic relationship between herpes simplex infection and recurrent *Aphthous stomatitis*.

Etiology

- The causative agent is a pleomorphic, transitional L - form of an α hemolytic streptococcus, *Streptococcus sanguis*.
- Immunologic abnormalities.
 - Result of an autoimmune response of the oral epithelium.
 - Local immune response against an antigenically altered mucosa → disease is the result of diffusion of bacterial toxins, foods and other substances acting as allergens or haptens which initiate an immune response.
 - An altered immune response which is directed against both the nonpathogenic oral flora and the host oral tissue.
- Iron, vitamin B₁₂ or folic acid deficiency allows the expression of an unrelated underlying tendency to ulceration and that the deficiency itself does not play a primary role.

Precipitating Factors

- Trauma
 - Self inflicted bites
 - Oral surgical procedures
 - Tooth brushing
 - Dental procedures

- Needle injections
 - Dental trauma
- Endocrine conditions
 - Menstruation
 - Pregnancy
 - Menarche
 - Menopause
- Psychic factors
 - Acute psychologic problem.
- Allergic factors
 - Asthma
 - Hay fever
 - Food or drug allergies.

Classification

- Based upon the clinical manifestations, recurrent aphthous stomatitis is classified into.
 - Recurrent aphthous minor (“Canker” sore).
 - Recurrent aphthous major (Sutton’s disease or periaadenitis mucosa necrotica recurrens).
 - Recurrent herpetiform ulcerations.
 - Recurrent ulcers associated with Behcet’s syndrome.

Clinical Features

- Recurrent aphthous minor.
 - Common between 10-30 years and predominantly in females.
 - Remarkable familial tendency.
 - Frequency of outbreaks of the aphthae varies.
 - Onset is manifested by the occurrence of one or more small nodules; generalized edema of the oral cavity, especially the tongue; paresthesia, malaise, low-grade fever; localized lymphadenopathy and vesicle-like lesion containing mucus.
- The aphthous ulcer begins as a single or multiple superficial erosion covered by a gray membrane, has a well-circumscribed margin surrounded by an erythematous halo.
 - Very painful.
 - The ulcer does not begin with a vesicle formation as in the case of herpes simplex infection.

- Common sites of occurrence are the buccal and lingual mucosa, buccal and lingual sulci, tongue, soft palate, pharynx and gingiva, all locations of labial mucosa not bound to periosteum.
 - Persist for 7-14 days and heal gradually with little or no scar formation.
- Recurrent aphthous major.
 - A female predilection.
 - Large, painful ulcers on the lips, cheeks, tongue, soft palate and fauces (one to ten in number).
 - Ulcers persist for up to 6 weeks and leave a scar upon healing.
 - Patient with severe major aphthae may also show similar lesions of the vagina or penis, rectum and larynx, with associated rheumatoid arthritis or conjunctivitis.
- Recurrent herpetiform ulcers.
 - Characterized by crops of multiple small, shallow ulcers.
 - Occur at any site in the oral cavity.
 - lesions begin as small pinhead sized erosions that gradually enlarge and coalesce.
 - More painful.
 - Lesions are present almost continuously for 1- 3 years with short remissions.
 - Immediate but temporary relief from symptoms with the use of 2 percent tetracycline mouthwash.

Histologic Features

- Oral mucous membrane exhibits a fibropurulent membrane covering the ulcerated area, with occasional superficial colonies of microorganisms.
- Connective tissue beneath the ulcer shows intense inflammatory cell infiltration, with tissue necrosis near the surface of the lesion, neutrophils below the ulcer and lymphocytes, adjacent to this.
- Margins may show epithelial proliferation.
- Accessory salivary gland tissue present in the area of aphthae may exhibit focal periductal and perialveolar fibrosis, ductal ectasia and mild chronic inflammation.
- Cytologic smearing reveals characteristic change in the nuclei of epithelial cells, "Anitschkow cells", which are cells with

elongated nuclei containing a linear bar of chromatin with radiating processes of chromatin extending towards the nuclear membrane.

Treatment

- Tetracycline mouthwash (250 mg per 5 ml) - 4 times a day for 5 - 7 days.
- 1.5 percent cortisone acetate.
- Hydrocortisone acetate - antibiotic lozenges.
- Chemical cautery (only pain reduction).
- Levamisole.
- Broad - spectrum antibiotics.
- Antiseptic (silver nitrate, gentian violet).
- Diet supplementation (Vit B₁₂, folic acid, iron, zinc sulfate)
- Symptomatic treatment (Xylocaine, silver nitrate, etc.)

BEHCET'S SYNDROME***Etiology***

- Behcet's syndrome has an immunologic basis because of strong associations with certain HLA types. The disorder appears to be an immunodysregulation that may be primary or secondary to one or more triggers, which include bacteria (especially streptococci), viruses, pesticides and heavy metals.

Clinical Features

- Occurs between 10-45 years of age.
- More common in males.
- Characterized chiefly by - Oral and genital ulcerations, ocular lesions, skin lesions.
- First manifestation is the oral/genital lesions.
- Oral lesions are very painful, occurs in crops similar to recurrent aphthous ulcers. The individual lesions may vary in size have ragged borders, surrounded a gray or yellow exudates. Most commonly involve the soft palate and oropharynx.
- The genital ulcers are small and present on the vulva, vagina, glans penis, scrotum and perianal areas. These lesions recur less frequently, are deeper and tend to heal with scarring.

- The ocular lesions begin with photophobia and irritation and the common manifestations are recurrent uveitis, hypopyon, iridocyclitis and conjunctivitis. Severe pain and blindness may result.
- Common cutaneous lesions include erythematous papules, pustules, vesicles, pyoderma, folliculitis, acneiform eruptions and erythema nodosum - like lesions. Most important skin manifestation is the presence of positive “pathergy”, (one or two days after injection of an inert substance, a tuberculin - like skin reaction or sterile pustule develops) is unique to Behcet’s syndrome.
- Arthralgia, thrombophlebitis, central nervous system involvement (paralysis and severe dementia), cardiovascular, pulmonary, gastrointestinal, renal complications may occur.

Histologic Features

- Most frequently seen pattern is leukocytoclastic vasculitis.
- The ulceration is similar to that seen in aphthous stomatitis, but the blood vessels classically demonstrate intramural invasion by neutrophils, karyorrhexis of neutrophils, extravasation of RBC’s and fibrinoid necrosis of the vessel walls.
- Endothelial proliferation is present.

Laboratory Findings

- Hypergammaglobulinemia.
- Leukocytosis with eosinophilia.
- Elevated ESR.

Diagnosis

- To differentiate Behçet’s syndrome from recurrent aphthous stomatitis, the following should be present :
- At least two of the classic triad of the disease must be present. Recurrent oral ulcers, recurrent genital ulcers and ocular inflammation.

Treatment and Prognosis

- Treatment is symptomatic and supportive

- Oral lesions → Tetracycline - containing rinse
 Topical corticosteroid
 Thalidomide (if resistant to steroids)
- Genital lesions → Topical corticosteroid
- Ocular lesions → Azathioprine and cyclosporine
- Skin lesions and → colchicines
arthralgia
- May undergo spontaneous remission, may progress to serious complications and even result in death.

REITER'S SYNDROME

- Reiter's syndrome is an uncommon disease that most likely represents an immunologically mediated condition. The disorder may be triggered by any one or several infectious agents in a genetically susceptible person.

Clinical Features

- Common in young adult men.
- The syndrome usually develops 1-4 weeks after an episode of dysentery or venereal disease.
- The typical tetrad of manifestations include.
 - Nongonococcal urethritis.
 - Arthritis
 - Conjunctivitis
 - Mucocutaneous lesions.
- Urethritis is the first sign and the urethral discharge is associated with an itching and burning sensation.
- Conjunctivitis is usually mild.
- Arthritis usually affects the joints of the lower extremities.
- Skin lesions have the characteristic manifestation on the glans penis (Balanitis circinata), which appears as well circumscribed erythematous erosions with a scalloped, whitish linear boundary. Other skin lesions consist of red or yellow keratotic macules or papules which eventually desquamate.

Oral Manifestations

- Oral lesions appear as painless, red, slightly elevated areas, granules or vesicular with a white circinate border on the buccal mucosa, lips and gingiva.
- Palatal lesions appear as small bright red purpuric spots which darken and coalesce.
- Tongue lesion are similar to that of geographic tongue.

Histologic Features

- Hyperparakeratosis, acanthosis and PMN leukocyte infiltration of epithelium, with microabscess formation.
- Lymphocyte and plasma cell infiltration of connective tissue.

Laboratory Findings

- Mild leukocytosis
- Elevated ESR
- Pyuria.

Treatment and Prognosis

- May undergo spontaneous remission.
- Can be treated by antibiotics and corticosteroids.

HERPANGINA

(Aphthous pharyngitis; vesicular pharyngitis)

- Herpangina is a specific viral infection caused by coxsackie group A viruses.

Clinical Features

- Most commonly seen in young children and incubation period is 2-10 days.
- Occurs in sporadic outbreaks and transmitted through contact.
- Many children harbour the virus in summer without exhibiting any clinical manifestation of the disease.
- Mild and of short duration.
- Onset occurs with sore throat, low-grade fever, headache, vomiting, prostration and abdominal pain.

- Small ulcers with a gray base and inflamed periphery develop on the anterior faucial pillars, hard and soft palates, posterior pharyngeal wall, buccal mucosa and tongue.
- Dysphagia may occur.
- Heal within a few days to a week.
- A permanent immunity to the infecting strain usually develops rapidly and most adults have neutralizing antibodies.

Treatment

- Self limiting disease.
- Complications include.
 - Acute parotitis
 - Meningitis
 - Hemolytic anemia
 - Hemorrhagic diathesis.

ACUTE LYMPHONODULAR PHARYNGITIS

- Acute lymphonodular pharyngitis is an acute febrile disease caused by coxsackie virus A10.

Clinical Features

- Incubation period is 4-7 days.
- Children and young adults are commonly affected.
- Crowding and poor hygiene aid in spread of infection.
- Characterized by sore throat, fever, mild headache and anorexia.
- The characteristic lesion consist of raised, discrete, whitish to yellowish solid papules surrounded by a narrow zone of erythema.
- The lesion represent hyperplastic lymphoid aggregates and resolve without vesiculation or ulceration.
- Commonly involved are the uvula, soft palate, anterior pillars and posterior oropharynx.
- Oral lesions may resolve within 6-10 days, although a residual ring of fading erythema may be present for several days.

Histologic Features

- Papular lesions consist of densely packed nodules of lymphocytes.

- Overlying epithelium may show either intranuclear or cytoplasmic inclusion bodies.

Treatment

- Self-limiting disease.

HAND, FOOT AND MOUTH DISEASE

- Hand, foot and mouth disease is an epidemic infection usually caused by coxsackievirus A16 but may also be due to other coxsackie A or B strains.

Clinical Features

- Common in young children.
- Characterized by the appearance of maculopapular, exanthematous and vesicular lesions of the skin affecting primarily the borders of the palms and soles and the ventral surfaces and sides of the fingers and toes, rarely, the buttocks may be involved. The skin lesion heal without crusting scarring.
- Anorexia, low grade fever, coryza and rarely lymphadenopathy, diarrhea, nausea and vomiting are present.
- The oral lesions are small, multiple vesicular and ulcerative lesions. A sore mouth with refusal to eat is the most common finding in this disease.
- Common sites for oral lesions are the hard palate, tongue and buccal mucosa.
 - Tongue becomes red and edematous.
 - Ulcerations resolve within 1 week.

Laboratory Findings

- Intracytoplasmic viral inclusions can be demonstrated in vesicular scrapings of the lesion.
- Remarkable rise in acute or convalescent serum antibody titer to coxsackie A16.

Histologic Feature

- The areas of affected epithelium exhibit intracellular edema, which leads to extensive spongiosis and formation of an intra-

epithelial vesicle. The vesicle enlarges and ruptures through the epithelial basal layer, with the formation of a subepithelial vesicle, epithelial necrosis and ulceration follows.

Treatment

- No specific treatment. Regresses within 1 or 2 weeks.

Differential Diagnosis

- There is similarity in clinical appearance between oral lesions of hand foot and mouth disease and other conditions which include.
 - Herpetic gingivostomatitis.
 - Herpangina.
 - Erythema multiforme.
 - Recurrent aphthous ulcers.
 - Animal foot and mouth disease.

MEASLES

(Rubeola; morbilli)

- Measles is an acute, contagious, dermatropic viral infection produced by a paramyxovirus.
- Affect primarily children and occurs in an epidemic form.
- Spread of infection occurs by either direct contact with an affected person or by droplet infection, the portal of entry being the respiratory tract.

Clinical Features

- Incubation period is 8-10 days.
- Prodromal symptoms include fever, malaise, coryza, cough, conjunctivitis, photophobia, lacrimation.
- The exanthematous rash follows after a few days and begins on the face, in the hair line and behind the ears and then spreads to the neck chest, back and extremities.
- The lesion appears as tiny, red macules or papules which enlarge and coalesce to form blotchy discolored, irregular lesions which blanch upon pressure and clears in four or five days in a similar downward progression and is replaced by a brown pigmentary staining.

Oral Manifestations

- The oral lesions are prodromal, occurring 2-3 days before the skin lesions and are pathognomonic of this disease, known as “Koplik’s spots”.
- These spots are small, irregularly shaped flecks which appear as bluish white specks surrounded by a bright red margin.
- Commonly occur on the buccal mucosa.
- These spots represent foci of epithelial necrosis and have been described as “grains of salt” on a red background.
- In case of severe malnutrition, candidiasis, acute necrotizing ulcerative mucositis and necrotizing stomatitis may also occur.
- Palatal and pharyngeal petechiae, congestion, swelling and focal ulceration of the gingiva, palate and throat may occur.

Complications

- Measles lowers of general body resistance and hence may lead to complications.
 - Bronchial pneumonia
 - Encephalitis
 - Otitis media
 - NOMA.
- Measles also has an immunosuppressive effect through impairment of cell-mediated immunity, which may lead to →
 - Delay in wound healing.
 - Induction of remission of leukemia and Hodgkin’s disease.
- Severe measles in early childhood can affect odontogenesis and result in pitted enamel hypoplasia of the developing permanent teeth.

Treatment and Prognosis

- Best treatment is MMR vaccine.
- In healthy patients, fluids and non-aspirin antipyretics may be used for symptomatic relief.
- In case of immunocompromised patients ribavirin, immunoglobulin, interferon and vitamin A can be used.

RUBELLA

(German measles)

- Rubella is a mild viral infection produced by a Togavirus.
- The virus is contacted through respiratory droplets.

Clinical Features

- Incubation period is from 14 -21 days.
- The infected patients are contagious from 1 week before the exanthema to 5 days after the development of rash.
- Infants with congenital infection may release the viruses for up to 1 year.
- Prodromal symptoms include fever, headache, malaise, anorexia, myalgia, mild conjunctivitis, coryza, cough, pharyngitis and lymphadenopathy (may persist for weeks and involves the sub-occipital, post auricular and cervical chains).
- The exanthematous rash first begins on the face and neck. The rash forms discrete pink macules then papules and then fades with flaky desquamation. The rash fades as it spreads and exhibits facial clearing before the completion of spread into the lower body areas.
- Oral lesions, known as "Forchheimer's sign" are small, discrete, dark-red papules that develop on the soft palate and extends onto the hard palate. This characteristic exanthema arises along with the rash and does not last more than 12 - 14 hours.
- Palatal petechiae are also present.
- The risk of congenital rubella is more when the disease occurs in the first trimester of pregnancy. The classic triad of congenital rubella syndrome consists of:
 - Deafness
 - Heart disease
 - Cataracts.
- Developmental defects, including enamel hypoplasia, high caries incidence and delayed eruption of deciduous teeth may also occur if the disease occurs during pregnancy.

Complications

- Less commonly, late - emerging complications include.
 - Encephalopathy
 - Mental retardation
 - Diabetes mellitus
 - Thyroid disorders.

Diagnosis

- Serologic analysis.

Treatment and Prognosis

- Non-aspirin antipyretics and anti-pruritic medications may provide symptomatic relief.
- Passive immunity may be obtained by administration of human rubella immunoglobulin. If it is given within a few days of exposure, the severity of the infections may be reduced.

SMALL POX

(Variola)

- Small pox is an acute viral disease, which was epidemic in nature and very fatal before the discovery of vaccination.

Clinical Features

- Incubation period is 7-10 days.
- Manifests with high fever, nausea, vomiting, chills and headache, patient is extremely ill and comatose in this period.
- The skin lesions as small macules and papules appearing first on the face and then spreading rapidly to the rest of the body. The papules develop into vesicles which then become pustules. The pustules are small, elevated and yellowish green with an inflamed border, which gets infected secondarily and become hemorrhagic.
- Desquamation occurs as healing begins.
- Common complication is severe pitting or pocking of the skin as a result of pustule formation.

- Oral lesions include multiple vesicle formation, which rupture and form nonspecific ulcers.
- Tongue is swollen and painful making swallowing difficult.

Complication

- Complications occur due to secondary infection, which include.
 - Abscess formation with septicemia
 - Respiratory infection
 - Erysipelas
 - Eye and ear infection.

CHICKEN POX

(Varicella)

- Chicken pox is an acute viral disease caused by varicella zoster virus (VZV; HHV - 3). Chicken pox represent the primary infection with the VZV; latency ensures and recurrence is possible as herpes zoster.
- Spreads through air droplets or direct contact with active lesions and portal of entry is respiratory tract.

Clinical Features

- Symptomatic phase of VZV infection begins with malaise, pharyngitis and rhinitis followed by → Maculopapular or vesicular eruptions of the skin and low grade fever.
- The lesion begins on the face and trunk followed by involvement of the extremities. Each lesion rapidly progresses through stages of erythema, vesicle, pustule and hardened crust and heal by desquamation.
- The early vesicular stage is most characteristic of the lesion. The centrally located vesicle is surrounded by a zone of erythema and described as “a dew drop on a rose petal”.
- The lesion occur in successive crops over 3 - 4 days and lesions in various stages are present at the same time.
- Oral lesions precede the skin lesions.
- Buccal mucosa and palate are commonly involved.

- The oral lesions are initially slightly raised vesicles with a surrounding erythema which rupture to form small eroded ulcers with red margin, resembling aphthous lesions. But, these lesions are not painful.

Complication

- Encephalitis
- Pneumonia
- Reye's syndrome
- Spontaneous abortion or congenital defect can occur if the disease occurs early during pregnancy.
- In immunocompromised persons, extensive cutaneous involvement along with high fever, hepatitis, pneumonitis, pancreatitis, gastrointestinal obstruction and encephalitis, may also occur.

Histologic Features

- Acantholysis, with formation of numerous free-floating Tzanck cells, which exhibit nuclear margination of chromatin and multinucleation.

Treatment and Prognosis

- Acyclovir
- Varicella-zoster immune globulin (VZIG), in immunocompromised persons.

MOLLUSCUM CONTAGIOSUM

- Molluscum contagiosum is caused by a virus of the pox group.
- Only occur on skin and mucosal surfaces.
- Considered tumor like due to the typical localized epithelial proliferation caused by the virus.

Clinical Features

- Common in children and young adults.
- Single or multiple discrete elevated nodules occurs on the arms, legs, trunk, face and particularly eyelids.
- Sexually transmitted or spread by autonoctulation.

- Incubation period is 14 - 50 days.
- Lesions are hemispheric in shape with a central umbilication which may be keratinized.
- Oral lesions may occur on the lips, tongue and buccal mucosa.

Histologic Features

- Thickening and downgrowth of the epithelium with the formation of large eosinophilic intracytoplasmic inclusion bodies known as "Henderson-Patterson inclusions" or " Molluscum bodies".

Treatment

- Surgical excision or topical application of podophyllin or cantharidin.

HERPES ZOSTER

(Shingles; zona)

- Herpes-zoster is an acute infectious viral disease caused by reactivation of the latent VZV which had been acquired during a previous attack of chicken pox.
- Predisposing factors for reactivation include.
 - Immunosuppression
 - Treatment with cytotoxic drugs
 - Radiation
 - Malignancies
 - Old age
 - Alcohol abuse
 - Dental manipulation.

Clinical Features

- Begins with pain in the area of epithelium innervated by the affected sensory nerve, (since the VZV is transported up the sensory nerves and establish latency in the dorsal spinal ganglia) usually unilaterally.
- Prodromal symptoms include fever, malaise and headache.
- The involved skin shows a cluster of vesicles set on an erythematous base within 3-4 days, the vesicles become pustular

and ulcerate, with crusts developing after 7- 10 days. The lesions tend to follow the path of the affected nerve and terminate at the midline.

- Resolves with 2-3 weeks, although secondary infection may slow the healing.
- There may be recurrences in the absence of vesiculation of the skin or mucosa, which is known as zoster sine herpete, characterized by severe pain of abrupt onset and hyperesthesia over a specific dermatome.
- Pain lasting longer than 1 month after an episode of herpes zoster is called post herpetic neuralgia.
- A special form of zoster infection of the geniculate ganglion, with involvement of the external ear and oral mucosa is known as James Ramsay Hunt's syndrome and is characterized by
 - Facial paralysis.
 - Pain of the external auditory meatus and pinna of the ear.
 - Vesicular eruptions of the oral cavity and oropharynx.
 - Hoarseness
 - Tinnitus
 - Vertigo.
- Occur involvement may arise from direct viral-mediated epithelial damage, neuropathy, immune-mediated damage or secondary vasculopathy.
- Tip of the nose may be involved when the nasociliary branch of the fifth cranial nerve is affected.
- Oral lesions occur with trigeminal nerve involvement and painful vesicles appear on the buccal mucosa, tongue, uvula, pharynx and larynx.
- Involvement of the maxilla is associated with devitalization of the teeth in the affected area.

Diagnosis

- Diagnosis is mainly from the clinical presentation.
- Other diagnostic methods include.
 - Viral culture
 - Cytologic smears
 - Direct staining of cytologic smears with fluorescent monoclonal antibodies for VZV.

Treatment and Prognosis

- High doses of acyclovir can decrease the duration of the exanthema and severity of the pain.
- Corticosteroids may be used to minimize the associated neuralgia that may arise.

MUMPS***(Epidemic parotitis)***

- Mumps is an acute contagious viral infection caused by a paramyxovirus, primarily affecting the salivary glands.
- The mumps virus can be transmitted through urine, saliva or respiratory droplets.
- Patients are contagious from 1 day before the clinical appearance of infection to 14 days after its clinical resolution.

Clinical Features

- Prodromal symptoms include low-grade fever, headache, malaise, anorexia, myalgia and pain below the ear.
- The parotid gland is involved frequently, but the sublingual and submandibular glands may also be affected.
- Discomfort and swelling develop in the tissue surrounding the lower half of the external ear and extending down along the posterior inferior border of the mandible.
- Pain is most intense during the period of maximal enlargement.
- Chewing movements of the jaw or eating saliva-stimulating foods tends to increase the pain.
- Enlargement of gland begins on one side and is followed by contralateral glandular changes within few days.
- Epididymo-orchitis occurs in males, the testicles exhibit rapid swelling with significant pain and tenderness.
- Oophoritis occurs in females.
- Meningo encephalitis, hearing loss, pancreatitis, arthritis, carditis, and decreased renal function may also occurs as complications.
- Frequent oral manifestation is redness and enlargement of Whartons and Stensen's salivary gland duct openings.
- Involvement of sub lingual gland may produce bilateral enlargements of the floor of the mouth.

Diagnosis

- Saliva, urine or cerebrospinal fluid culture.
- Acute and convalescent mumps-specific antibody measurements.

Treatment and Prognosis

- Treatment of mumps is palliative.
- As with measles and rubella, the best results come from prior vaccination, thus preventing infection.

INFECTIOUS MONONUCLEOSIS

(Glandular fever, kissing disease)

- Infectious mononucleosis is a symptomatic disease caused by Epstein - Barr virus (EBV, HHV - 4), which also causes Burkitt's African jaw lymphoma, nasopharyngeal carcinoma and lymphoblastic leukemia.
- Infection usually occurs by intimate contact once exposed, EBV remain in the host for life.
- Children usually become infected through contaminated saliva on fingers or toys. Adults become infected through direct salivary transfer, through shared straws or kissing and hence also known as "Kissing disease".
- Increasing number of opportunistic infections or neoplastic processes.

Clinical Features

- Asymptomatic in children mostly. If symptoms occur, may include fever, lymphadenopathy, pharyngitis, hepatosplenomegaly, rhinitis or cough.
- In young adults, prodromal symptoms include fatigue, malaise, fever, sore throat, headache, chills and vomiting.
- Prominent lymphadenopathy presents as enlarged, symmetric and tender nodes involving the posterior and anterior cervical chains. Nodes of the axilla and groin may also be involved.
- Involvement of the parotid lymphoid tissue may be associated with facial nerve palsy.

- Oropharyngeal tonsillar enlargement, with diffuse surface exudates and secondary tonsillar abscesses.
- Petechiae on the hard or soft palate, which are transient.
- ANUG, ANUG - like pericoronitis, acute necrotizing ulcerative mucositis.
- Edema of the soft palate and uvula.

Diagnosis

- Lymphocytosis, with more atypical forms.
- Monospot test → Presence of Paul - Bunnell heterophil antibody. (Normal titre of agglutinin and hemagglutinins, in human blood against sheep RBC's may arise to 1:4096 as compared to normal of 1:8).
- Indirect immunofluorescence test.

Treatment and Prognosis

- Resolves within 4 - 6 weeks.
- Antibiotics and corticosteroids may be used.

ACQUIRED IMMUNODEFICIENCY SYNDROME (HIV; AIDS)

- AIDS is caused by human immunodeficiency virus (HIV), a member of Retroviridae family. Within the retroviridae family HIV is classified as lentivirus, which encode for gene products, e.g. gag, pol, env and accessory proteins, which play an important role in pathogenesis.

Modes of Transmission

- HIV is found in most bodily fluids of infected persons, including serum, blood, saliva, semen, tears, urine, breast milk, ear secretion and vaginal secretions.
- The most frequent routes of transmission are
 - Sexual contact.
 - Parenteral exposure to blood.
 - Transmission from mother to fetus during the perinatal period.
 - Artificial insemination.

- Breast-feeding from infected mothers.
- Organ transplantation.
- Intravenous drug abusers.

Pathogenesis

- The primary target cell of HIV is the CD4⁺ helper T- lymphocyte. The DNA of HIV is incorporated into the DNA of the lymphocyte and is thus present for the life of the cell. In persons with HIV infection, antibodies are developed, but are not protective.
- The virus may remain silent, cause cell death or produce fusion of the cells into syncytia, which may not function properly.
- A subsequent decrease in T- helper cell number occurs, with a resultant loss in immune function. The normal response to viruses, fungi and encapsulated bacteria is diminished.
- On introduction of the HIV, an indefinite percentage of those infected develop an acute self-limited viral syndrome. This is followed by an asymptomatic stage which averages 8-10 years. Then the final symptomatic stage develops.

Clinical Features

Acute Viral Syndrome

- Develops within 1-6 weeks after exposure.
- Symptoms include generalized lymphadenopathy, sore throat, fever, maculopapular rash, headache, myalgia, arthralgia, diarrhea, photophobia, peripheral neuropathies.
- Oral changes include mucosal erythema and focal ulceration.
- Clears within a few weeks.

Asymptomatic Stage

- Persistent generalized lymphadenopathy.
- AIDS - related complex (ARC) which includes chronic fever, weight loss, diarrhea, oral candidiasis, herpes zoster and / or hairy leukoplakia.

Symptomatic Stage

- Highly variable and is affected by a person's prior exposure to a number of chronic infections.

- Signs and symptoms of ARC may be present.
- Most common features leading to diagnosis is pneumonia caused by *Pneumocystic carinii*.
- Other infections of diagnostic significance include disseminated CMV infection, severe herpes simplex virus infection, atypical mycobacterial infection, *Cryptococcal meningitis* and CNS toxoplasmosis.
- Persistent diarrhea of bacterial or protozoal origin.
- Most common manifestation of neurologic dysfunction is a progressive encephalopathy known as AIDS - dementia complex.
- Most common neoplastic processes include Kaposi's sarcoma and Non-Hodgkin's lymphoma.

Diagnosis

- Confirmation of HIV infection is made by viral culture or by detection of HIV antibodies or antigens.
- Viral-antibody detection.
- Viral-antigen detection.
- Polymerase chain reaction (PCR).

Treatment and Prognosis

- AIDS is considered fatal.
- Survival times are increased as a result of earlier diagnosis, improved therapy of infections and malignancies and therapy with zidovudine (AZT).

Mycotic Infections of Oral Cavity

- Mycotic infection means fungal infection. Nowadays fungal infection is more common than bacterial. So to distinguish between mycotic infection and other diseases the epidemiology, pathogenesis, immunology, diagnosis and treatment plan should be studied in detail.
- Infection caused by fungus- Mycosis.
- Humans have high level of immunity to fungi. This resistance is due to:
 - Fatty acid content of the skin.
 - pH of the skin and mucosal surface.
 - Epithelial turnover.
 - Normal flora.
 - Cilia of the respiratory tract.

CLASSIFICATION

- According to tissue levels initially colonized.
- Superficial mycoses.
- Cutaneous mycoses.
- Subcutaneous mycoses.
- Systemic mycoses.
- Opportunistic mycoses.

Superficial Mycoses

- Infection is limited to the outermost layers of the skin and hair.
- Pityriasis versicolor.
- Tinea nigra.
- Black piedra.
- White piedra.

Cutaneous Mycoses

- Infection that extends deeper into the epidermis, as well as invasive hair and nail diseases.
- Limited to keratinized layer of skin, hair and nail diseases
Causative agent- Dermatophytes disease- Ringworm/Tinea.
For example: *Tinea capitis*, *Tinea manus*, *Tinea pedis*.

Subcutaneous Mycoses

- Infection involving the dermis, subcutaneous, muscles and fascia exhibits chronic insidious growth pattern.
For example: Sporotrichosis
- Chromoblastomycosis.
- Mycetoma.
- Rhinosporidiosis.

Systemic Mycoses

- Infections that originate primarily in the lung and they spread to many organ systems.
- Usually caused by fungi of soil and is acquired by inhalation.
For example: Cryptococcus.

Blastomycosis

- Paracoccidioidomycosis.
- Histoplasmosis.
- Systemic candidiasis.

Opportunistic Mycosis

- Infections in patients with immune deficiencies who would otherwise not be infected.

- Occurs in AIDS, Diabetes mellitus and Immunosuppressive therapy.
For example: Candidiasis.
 - Aspergilloses.
 - Zygomycoses.

Oral Fungal Infections

- Oral candidiasis
- Histoplasmosis
- Blastomycosis
- Paracoccidioidomycosis
- Coccidioidomycosis
- Cryptococcosis
- Zygomycosis
- Aspergillosis
- Taxoplasmosis.

Oral Candidiasis

- Oral candidiasis is a relatively common well known opportunistic mycotic infection in man.
- Species of Genus *Candida* - causative fungi.
- In 1923 *Berkhout* coined the term.
- *Candida*- TOGACANDIDA (White robe worn by candidates of Roman senate).
- It is a relatively frequent mycosis of worldwide distribution.
- Genus *Candida*, 150-200 species.
- Eight species- pathogenic, e.g. *C albicans*, *C tropicalis*, *C glabrata*, *C krusei*, *C parapsilosis*, *C famata*, *C rugosa*, *C pseudotropicalis*.
- *Candida albicans*—frequently isolated species.
- Albicans-Albicare (to whiten).
- 'A disease of the diseased'—because of its frequency in association with AIDS.

Predisposing Factors

- Local host factors
- Systemic factors
- Iatrogenic factors.

Local Host Factors

Mucosal Barriers

- Exogenous epithelial changes.
 - Trauma
 - Local occlusion
 - Maceration.
- Endogenous epithelial changes.
 - Atrophy
 - Hyperplasia
 - Dysplasia.

Saliva

- Qualitative changes.
 - Xerostomia
 - Sjögren's syndrome
 - Radiotherapy.
- Quantitative changes.
 - pH
 - Glucose concentration.

High Carbohydrate Content Commensal Flora

- Systemic factors.

Altered Physiological State

- Infancy
- Old age
- Pregnancy.

Altered States

Altered Hormonal State

- Diabetes
- Hypothyroidism
- Hypoadrenocretinism.

Systemic Factors

Altered Nutritional State

- Hypovitaminosis
- Iron deficiency
- Malnutrition.

Altered Immune Mechanism

- Decreased number of phagocytes.
- Intrinsic defects in immune cells.
- Defects in cell mediated immunity.

Latrogenic Factors

- Antibiotics
- Corticosteroids
- Contraceptives
- Immunosuppressive drugs.

Miscellaneous Factors

- Smoking
- Secretory status.

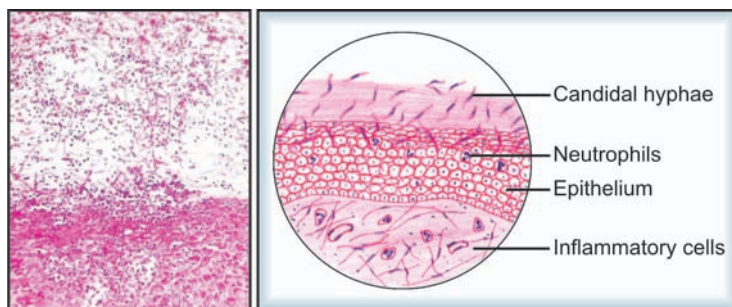


Fig. 9.1: Candidiasis

Classification of Oral Candidiasis

- Samaranayake and Nair - 1995.

Oral candidiasis

- Primary oral candidiasis
- Secondary oral candidiasis.

Primary Oral Candidiasis

Acute form

- Pseudomembranous.
- Erythematous.

Chronic form

- Hyperplastic
- Nodular
- Plaque like
- Pseudomembranous.

Candida associated lesions

- Denture stomatitis
- Angular cheilitis
- Median rhomboid glossitis.

Super infected with candida

- Leukoplakia
- Lichen planus
- Lupus erythematosus.

Secondary Oral Candidiasis

Oral manifestation of systemic mucocutaneous candidiasis

- Familial chronic mucocutaneous candidiasis.
- Diffuse chronic mucocutaneous candidiasis.
- HIV and AIDS.

Candida carriage in oral cavity

- Tony Axell, Samaranayake and Reichart (1997)
- Neonates - 45-65%
- Healthy adults - 30-45%
- People with dentures - 50-65%
- HIV and other immunosuppressive diseases - 90%

Pathogenesis of candidial organisms

- Krempel (1991)
- Pathogenesis varies in different biotypes and strains of Candida

- Enzymes
- Temperature and pH variations
- Adhesion of candida
- Sterols
- Oral flora
- Secretory status
- Switching phenomenon
- Pseudomembranous candidiasis (thrush).

Symptoms

- Mild burning sensation of oral mucosa
- Unpleasant taste
- Blisters
- Palate, buccal mucosa, dorsum of the tongue C/F
- Adherent white plaque which resemble cottage cheese or curdled milk on the oral mucosa
- White plaques—hyphae, yeast, desquamated epithelial cells and debris
- Plaques - scraping with tongue blade or dry gauze piece.
- Underlying epithelium appears normal/erythematous.
- If bleeding occurs—lichen planus/cancer chemotherapy.
- Also initiated by—Broad spectrum antibiotics.
- Immunodeficiency.

Erythematous Candidiasis

- Does not show white flecks.
- Acute atrophic candidiasis/ Antibiotic sore mouth.

Symptoms

- Mouth being scaled by hot beverages.
- Burning sensation.
- Bald appearance of the tongue C/F.
- Erythematous mucosa - Dorsum of tongue.
- Junction of soft and hard palate, angle of the mouth.
- Central papillary atrophy of the tongue/median rhomboid glossitis.

ANGULAR CHEILITIS (PERLECHE)

Symptoms

- Red, fissured lesions.
- Irritated raw feelings.

Site

- Angle of the mouth.
 - Idiopathic
 - Immunosuppression
 - Loss of vertical dimension.

DENTURE SORE MOUTH

Types

- Newton's type I—Localized simple inflammation/pin-point hyperemia.
- Newton's type II—Diffuse erythema involving a part/entire denture bearing area.
- Newton's type III—Inflammatory papillary hyperplasia involving central part of hard palate and alveolar ridge.

PAIN AND BURNING SENSATION

Clear demarcation seen between affected and normal mucosa.

Candida Associated Denture Stomatitis

Histopathology

- Hyphae are not seen in epithelium.
- Epithelial atrophy.
- Intraepithelial leukocyte infiltration.
- Acanthosis.

Candidial Leukoplakia

- Chronic hyperplastic candidiasis.
- Clinical features.
- Usually above 50 years.
- Men > women.

- Frequency in decreasing order commissures, cheeks, palate, tongue (dorsum) and lips.
- Homogenous/Speckled.
- Lesions cannot be wiped off.
- Multiple irregular, firm nodules on an erythematous velvety mucosal background.

HISTOPLASMOSIS

Clinical Features

- Causative organism—*Histoplasma capsulatum*.
- Airborne spores—Infects after inhalation.
- Expression of the disease = Quantity of spores inhaled.
- Most cases patient exposed to mild number of spores—Relatively healthy, either no symptoms or flu like illness for 1-2 weeks.
- Massive inhalation—Acute pneumonia.
- Serious sequelae includes—Fibrosing pulmonary infection, disseminated infection with bone marrow and adrenal involvement.
- Acute histoplasmosis.
- Patient exposed to low number of spores—Self limited pulmonary infection.
- High number of inhalation of spores—Fever, headache, myalgia, cough, anorexia chronic histoplasmosis.
- Less common compared to acute form.
- Primarily affects lungs.
- Clinically resembles tuberculosis—cough, weight loss fever, chest pain, weakness, hemoptysis, and fatigue.
- Chest radiograph—Upper lobe infiltrates and cavitations.
- Disseminated histoplasmosis.
- Even less common.
- Progressive spread of infection to extrapulmonary sites—Spleen, adrenal glands, liver, lymph nodes, CNS, oral mucosa and skin.
- Usually occurs in elderly, debilitated or immunocompromised patients.
- US association with AIDS is—2% .

Oral Manifestations

- Usually occurs with disseminated form.
- Nodules over the mucosa which undergoes ulcerations gingiva.
- Commonly infected sites—Tongue, palate and buccal mucosa.
- Lesion presents as solitary, painful ulceration of several weak duration.
- Some lesions may appear as erythematous or white with an irregular surface.

Diagnosis

- Culture, serology.
- Biopsy -Special stains such as.
(PAS, Grocott's-Gomori methenamine silver).

Histopathology

- Lesional tissue shows either a diffuse infiltrate of macrophages or more commonly collection of macrophages organized into granuloma.
- Multinucleated giant cells (Langerhans') are usually seen in granulomatous inflammation.
- Presence of characteristic 1-2 micron yeasts of *H capsulatum*.

BLASTOMYCOSIS

(Gilchrist's disease)

- Caused by dimorphic fungi—*Blastomyces dermatitidis*.
- Organism prefer rich moist soil where it develops as mould.
- Affects endemically in North American states.
- Prominent adult male predilection—9:1.
- Occurrence in immunocompromised patient is rare.

Clinical Features

- Acquired by inhalation of spores particularly after rain, spores reaches alveoli and grow as yeasts at body temperature.
- Begins as small red papules which gradually ↑ in size form abscesses or pustules and then ulcerate.
- Primary foci of infection is lungs.

- Spreads hematogenously and affects skin, bone, prostate, meninges, oropharynx and abdominal organs.

Acute Blastomycosis

- Resembles pneumonia.
- Regresses after treatment.

Chronic Blastomycosis

- Mimics Tuberculosis.
- Chest radiographs -Normal, does not show calcifications.
- Cutaneous lesions begin as erythematous nodules that enlarge and become verrucous or ulcerated.

Oral Manifestations

- Oral lesions result from extrapulmonary dissemination.
- Lesions may have an irregular, erythematous or white intact surfaces or they may appear as ulceration with irregular rolled borders.
- Usually associated with pain.
- Clinically they resemble squamous cell carcinoma.

Diagnosis

- Biopsy- PAS, Grocott's - Gomori methenamine silver H and E.
- Cytology-20 percent KOH.

Culture

- Most accurate method, but slow takes 3-4 weeks to demonstrate organism.
- DNA probe—Allows immediate identification of mycelium phase of hyphae.

Histopathology

- Typically shows mixture of acute inflammation and granulomatous inflammation surrounding variable numbers of yeasts.
- Organisms are 8-10 microns in diameter.
- Organisms appear as doubly refractile cell wall and broad attachment between the budding daughter cell and the parent cell.

PARACOCCIDIOIDOMYCOSIS

- South American Blastomycosis.
- Frequently found in peoples of South and Central America.
- Caused by *Paracoccidioides brasiliensis*.
- Distinct male predilection - 25:1.
- This difference is thought to be due to protective effect of female hormones.
(*B estradiol* inhibits the transformation of hyphae form to the pathogenic yeast form).

Clinical Features

- Affects middle aged groups.
- Initially appears as pulmonary infection.
- Self limiting.
- Usually spreads by hematogenous/lymphatic route.
- Extends - skin, mucous membrane and adrenal glands.
- Adrenal involvement results in Addison's disease oral manifestations.
- Usually appears as mulberry like ulcerations.
- Most commonly affects alveolar mucosa, gingiva and palate.
- Rarely involves lips, oropharynx and buccal mucosa.

Oral Lesion

- Involves more than one bite.

Diagnosis

- Culture
Biopsy – Grocott's - Gomori methenamine silver and H and E

Histopathology

- Microscopy reveals pseudoepitheliomatous hyperplasia in addition to ulcerations of surface epithelium.
- Collection of epithelioid macrophages and multinucleated giant cells.
- Yeast cells are scattered—larger in size up to 30 microns.
- Organisms often show multiple daughter buds on parent cell resulting in appearance resembling 'Mickey Mouse Ears' OR 'Mariner's Wheel'.

COCCIDIOIDOMYCOSIS

- Coccidioidomycosis—Causative fungi.
- Confined to the western hemisphere and endemic throughout the desert region of US and Mexico states.
- Dimorphic—Appears as moulds in natural environment and yeast in infected host.

Clinical Features

- Most infection are asymptomatic.
- 40 percent of the patient experience flu like and pulmonary infections, symptoms last for several weeks with spontaneous resolution.
- Occasionally immune response may trigger a hypersensitive reaction that causes development of *Erythema multiforme* or *Erythema nodosum*.
- Lesions characterized by multiple painful inflammatory nodules in the subcutaneous connective tissue.
- Hypersensitivity reaction occurring to conjunction with coccidioidomycosis - Valley fever.
- Chronic form resembles Tuberculosis.
- Disseminated form spreads hematogenously -Skin, bones and joints, meninges.
- Oral lesions are usually uncommon.

Histopathology

- Biopsy reveals large 20-60 microns round spherules that may contain numerous endospores.

CRYPTOCOCCOSIS

- Relatively uncommon fungal disease—*Cryptococcus neoforms*.
- The disease has worldwide distribution because of its association with pigeon.
- Grows as yeast in both soil and infected host.
- Commonly affects immunocompromised patient.

Clinical Features

- Cough with mucoid expectoration, pleuritic pain
- Papular, nodular ulcerative lesions → skin
- Meningoencephalitis → associated brain infection.

Histopathology

- Generally shows granulomatous inflammatory response to the organism.
- Yeast appears as round to ovoid structure with 4-6 microns diameter surrounded by a clear halo that represents the capsule.

Treatment

Amphotericin-B.

ZYGOMYCOSIS

(Mucormycosis, phycomycosis)

- It is an opportunistic, frequently, fulminant fungal infection-saprobic organisms of the class Zygomycetes.

Causative Fungi

- *Z. absida*, *Z. mucor*, *Z. rhizomucor* and *Z. rhizopus*.
- Organisms grows in their natural state on variety of decaying organic materials.
- Spores are liberated in to air → inhalation → infection in man.
- Rhinocerabral form is the most relevant.
- They have been reported rarely in normal human flora.
- Especially seen in Insulin dependent uncontrolled diabetic patients with ketoacidosis.
- Also affects Immunocompromised patients .

Oral Manifestations

- Usually begins in maxillary antrum particularly in young adults, poorly controlled diabetes.
- Invasion of surrounding tissue leads to necrotizing ulceration of palate, blackish slough formation and exposure of bone.
- Low fever, local pain and tenderness over the sinus.
- Radiographic evidence - spotty destruction of walls (Paranasal air sinuses).
- Nasal congestion, bloody nasal discharge.
- When spreads to orbit causes blurred vision, proptosis and limitation of eye movements.
- Involvement of cranial vault - Fatal.

Histopathology

- Extensive necrosis with numerous large (6-30 microns), branching non-separate hyphae.
- Hyphae tend to branch at 90° angles.
- Hyphae are characterized by 'Crushed ribbon' appearance.
- Lesions involve blood vessels with thrombosis, hemorrhages, inflammatory response and tissue necrosis.

ASPERGILLOSIS

- It is a fungal disease characterized by noninvasive and invasive form.
- Two important species are *A. flavus* and *A. fumigatus*.
- *A. fumigatus* being responsible for 90 percent of cases.
- Noninvasive form usually affects normal host, appears as an allergic reaction.
- Invasive forms are more evident in immunocompromised patients.

Clinical Features

- Clinical manifestations vary depending on the host immune status and presence or absence of tissue damage.
- In normal host disease appears as an allergy affecting either sinuses or bronchopulmonary tract (allergic fungal sinusitis).
- Sometimes asthmatic attacks may be triggered by inhalation of spores.
- Low grade infections sometimes establish in maxillary sinus resulting in mass of fungal hyphae called as Aspergilloma.
- Occasionally the fungal mass under go dystrophic calcification—Anthrolith in the sinus.

Oral Manifestations

- Occurs in maxillary posterior segment.
- Portal entry to the infection may be through marginal gingiva/ gingival sulcus.
- Painful gingival ulcerations, peripherally the mucosa and the soft tissue develops diffuse swelling with a violaceous hue.
- If left untreated-extensive necrosis with yellow or black ulcers and facial swelling can be seen disseminated Aspergillosis.

- Occurs primarily in immunocompromised patients, particularly leukemia, AIDS.
- Patients exhibits—chest pain, fever.
- Hematogenous spread—spread to CNS, eye, skin, live, G.I. tract and thyroid gland.

Histopathology

- Lesions show varying numbers of branching, septate hyphae - 3-4 microns.
- Hyphae have a tendency to branch at acute angle and invades adjacent small blood vessels.
- Occlusion of the vessels often results in necrosis.

Treatment

- Antifungal drugs—Amphotericin-B.
- Mild conditions—Flucytocine, itraconazole and ketoconazole.
- Severe conditions—Anti-allergic drugs and corticosteroids.
- Allergic conditions—Surgical line of treatment.

Allergic and Immunologic Diseases of Oral Cavity

RECURRENT APHTHOUS STOMATITIS

Synonyms

- Aphthous ulcers
- Aphthae
- Canker sores
- Painful lesion present with solitary or multiple ulcers
- Recurring lesion.

Etiology

- Bacterial infection: (Alpha hemolytic streptococci) this organism is isolated from aphthous patients and when administered into the guinea pig produce lesions on skin and oral mucosa similar clinically and histologically to the aphthous lesions. When patient tested with *Streptococcus* vaccine, gives a positive delayed type of hypersensitivity.
- Immunologic abnormalities: IgG and IgM binding by epithelial cells of spinous cells of oral mucosa is identified in aphthous patients.
- Iron, Vitamin B₁₂ or Folic acid deficiency.

Precipitating Factors

- Trauma—Local trauma.
- Endocrine conditions—Premenstrual period.
- Psychologic factors.
- Allergic factors—Asthma, Hay fever, Drug allergies.

Classification

- Recurrent aphthous minor.
- Recurrent aphthous major.
- Recurrent herpetiformi.
- Ulcers associated with Behcet's syndrome.

(a) Recurrent Aphthous Minor

- Most common form.
- Women > men.
- Onset between 10 and 30 years.
- Show recurrence.
- 1 or 2 attacks per year may be seen.
- Small nodule formation.
- Prodromal symptoms like fever malaise and paresthesia, lymphadenopathy is seen.
- Formation of vesicles containing mucus.
- Rupture of vesicles.
- Formation of ulcers.
- They are covered by grey membrane.
- They have well circumscribed margins surrounded by an erythematous halo.
- Lesions are very painful.
- Their number may range from 1 to 100.
- Occurs on buccal mucosa, tongue, soft palate and, etc.
- They persist for 7-14 days.

(b) Recurrent Aphthous Major

- Severe form
- Large and painful
- Occur at frequent intervals
- Persist for 6 weeks.

- Scar upon healing.
- Females commonly affected.
- Seen on lips, tongue, cheeks and etc.

(c) Herpetiformi

- Multiple shallow ulcers up to 100 in number.
- Occurs at any site.
- Lesions are similar to herpetic lesions clinically.
- They differ histologically.
- Begin as small pin head sized erosions that gradually enlarge and coalesce.
- Present continuously for 1-3 years.
- Lab investigations showed negative culture for HSV.
- Cytologic smears fail to reveal the typical multinucleated epithelial giant cells found in herpetic lesions.
- Serologic tests were negative for antibodies to herpes virus.

Histologic Features

- Fibrinopurulent membrane covering the ulcerated areas.
- Microorganisms are present in this membrane.
- Intense inflammatory infiltrate.
- Necrosis of the tissue near the surface of the lesion.
- Polymorphonuclear neutrophils cells predominate.
- Epithelial proliferation is seen at the margins
- Cytological smears shows nuclear changes.
- These cells are called **ANITSCHOW** cells.
- They contain a linear bar of chromatin with radiating processes extending toward the nuclear membrane.
- These cells are also seen in pts. with sickle cell disease, megaloblastic anemia and iron deficiency anemia.

Treatment

- Immune enhancement
 - Levamisole
 - vaccine
- Immune suppression
 - Prednisolone
 - Triamsinolone acetonide

- Antibiotics
 - Tetracycline suspension
 - Chloramphenicol
- Antiseptics
 - Silver nitrate
 - Coagulating agent
- Diet supplementation
 - Vitamin B₁₂, folic acid
 - Iron
- Symptomatic treatment
 - Xylocaine, lignocaine
 - Silver nitrate
 - Benadryl.

BEHCET'S SYNDROME

- Initially thought to arise from pneumonia like organisms.
- Now it has shown to arise from autoimmune etiology.
- Occurs at an age of 30 years.
- More common in males.
- Diet supplementation
 - Vitamin B₁₂, folic acid
 - Iron
- Symptomatic treatment
 - Xylocaine, Lignocaine
 - Silver nitrate
 - Benadryl.

Shows Oral, Genital and Skin Lesions

- First oral and genital involvement.
- The lesions are painful.
- Occular lesions start as photophobia and irritation.
- Skin lesions are generally small pustules or papules on trunks limbs and genitalia.
- Cardiac and pulmonary involvement is also seen.

Histologic Features

- They show endothelial proliferation.
- Lab investigations shows hypergammaglobulinemia, leukocytosis, eosinophilia and there is increased ESR.

REITER'S SYNDROME

- Disease of unknown etiology.
- Clinically mimics gonorrhea.
- But urethral discharge is negative for gonorrhea.
- Bedsonia group of organisms are isolated.
- Seen in young adults.
- Men are commonly involved.
- Patient may present urethritis, conjunctivitis, and mucocutaneous lesions and arthritis.
- Urethral discharge is associated with itching and burning sensation.
- Arthritis is often bilateral.

Oral Manifestations

- Appear as elevated areas.
- Sometimes granular and vesicular.
- Palatal lesions appear as bright red purpuric spots which darken and coalesce.
- Lesions on tongue resembles geographic tongue.

Histologic Features

- They are not diagnostic.
- Lab findings shows leukocytosis and increased ESR.

Treatment

- Involves Antibiotics and Corticosteroids.

SARCOIDOSIS

- Multisystem granulomatous disease of unknown etiology characterized by the formation of uniform, discrete, compact and noncaseating epithelioid granulomas.
- Affects young adults.

- Patients present with hilar lymphadenopathy, pulmonary infiltration, skin, and eye lesions.
- Hence lesions commonly seen in lungs, skin, lymph nodes, salivary glands, spleen and bones.
- There is depression of delayed type of hypersensitivity which suggests impaired cell mediated immunity and raised abnormal serum immunoglobulin.
- Affects young and middle aged adults.
- Cutaneous lesions are also seen.
- They appear as multiple raised red patches.
- They grow slowly and do not ulcerate.
- Lymph node and salivary gland involvement will cause nodular enlargement.

Oral Manifestations

- Usually small papular nodules or plaques can be seen.

Histologic Features

- Resemble noncaseating nodules.
- Non-acid-fast bacilli can be demonstrated.
- Nests of epithelioid cells and multinucleated giant cells are seen.
- Lab findings shows an elevated levels of ACE in serum.

UVEOPAROTID FEVER

- Characterized by firm painless bilateral enlargement of parotid glands.
- Accompanied by inflammation of uveal tract of eyes.
- Sublingual, submaxillary and lacrimal glands are also involved.
- Chronic low grade fever with nausea, vomiting and xerostomia is seen.
- Unilateral or bilateral nerve involvement of 7th cranial nerve is seen.
- The signs and symptoms will disappear in time.

MIDLINE LETHAL GRANULOMA

Synonyms

- Malignant granuloma.
- Lethal granuloma.
- Midline lethal granulomatous ulcerations.

Many other diseases also manifest like these which are broadly classified as follows:

Infectious Diseases

(a) Bacterial

- Brucellosis
- Rhinosclerosis
- Leprosy
- Actinomycosis
- TB
- Syphilis.

(b) Fungal

- Histoplasmosis
- Candidiasis
- Blastomycosis.

(c) Parasitic

- Leishmaniasis
- Myiasis.

(d) Neoplastic

- Squamous cell carcinoma
- Rhabdomyosarcoma
- Conventional lymphoma.

Clinical Features

- Resembles serious infection.
- Characterized by idiopathic progressive destruction of nose, Paranasal sinuses, palate, face and pharynx.
- Clinically appear as ulcerations of palate or nasal septum.
- Preceded by feeling of stiffness in the nose.
- Bear resemblance to carcinoma.
- Persists for months to years.
- Involved bones undergo necrosis and sequestration.
- Proliferating sinus tracts may develop.
- Patient may even die if blood vessels are involved, causing

hemorrhage.

Histologic Features

- There is extensive necrosis.
- Inflammatory infiltrate.
- Repeated biopsies are necessary to rule out the carcinoma.

Treatment

- Involves corticosteroid therapy.

INFLAMMATORY DISEASES OF UNKNOWN ETIOLOGY

- Wegener's granulomatosis.
- Idiopathic midline destructive disease.

WEGENER'S GRANULOMATOSIS

- Involves vascular, renal and respiratory systems.
- Also involves nose, paranasal air sinuses, lower respiratory tract, gut, joints, nervous system and kidneys.
- Kidney involvement is the common cause for death.
- Caused by an immune reaction secondary to non-specific infection or unknown antigen.
- Occurs at the age of 4-5th decade of life.
- Males are more commonly affected.
- First characterized by rhinitis, sinusitis, otitis or ocular symptoms.
- Lungs also get involved.
- Cough and hemoptysis is seen.
- Glomerulonephritis causes uremia and renal failure.

Oral Manifestations

- Includes involvement of gingiva causes strawberry gingivitis.
- Starts at interdental papilla and periodontal ligament is involved as well as bone and causes mobility of teeth.

Histologic Features

- Inflammation around blood vessel is seen along with giant cells and necrosis.

Treatment

- Includes cytotoxic agents like cyclophosphamide, prednisone.

CHRONIC GRANULOMATOUS DISEASES

- Characterized by severe recurrent infection as a result of intracellular leukocyte enzymatic function with a decreased oxidative metabolism in which there is failure to destroy certain catalase positive microorganisms including staphylococci enteric bacilli and fungus.
- Clinically affects lymph nodes, lungs, liver, spleen, bone and skin.
- Later stages skin shows granuloma formation.

Oral Manifestations

- Include diffuse stomatitis with or without solitary or multiple ulcerations.
- Some patients show benign migratory glossitis.
- Enamel hypoplasia of permanent teeth also noted.

Histologic Features

- Granulomas with mononuclear histiocytes and multinucleated giant cells are seen.

ANGIOEDEMA

Synonyms

- Angioneurotic edema.
- Quenke's edema.
- Giant urticaria.
- Characterized by diffuse edematous swelling of soft tissues commonly involving the subcutaneous and submucosal connective tissues.
- When gastrointestinal tract and respiratory tract is involved, it leads to death.
- This is mainly due to alteration in vascular permeability.
- Since many patients of angioedema present with psychological problems, it is referred to as angioneurotic edema.

Causes

- Allergy due to mast cell degranulation.
- This leads to histamine release and the typical clinical manifestation is seen.
- This is caused by drugs, foods, plants, dust, and inhalants.

Clinical Features

- Involve lips, chin, eyes, legs, pharynx and larynx.
- Soft, nontender, edematous, swelling of rapid onset.
- This may be solitary or multiple.
- Some times hands, arms and genitals may involve.
- Itching precedes the swelling.
- The skin may be normal or pink in color.
- Perioral and periorbital edema is seen.
- The enlargement recedes in 24 to 72 hours.
- Involvement of respiratory system is dangerous.
- Gastrointestinal tract symptoms include continuous pain vomiting and watery diarrhea.

Treatment

- Elimination of etiological agents.

DRUG ALLERGY

Synonyms

- Drug idiosyncrasy
- Drug sensitivity
- Dermatitis
- Dermatitis medicamentosa
- Caused by exposure to drugs
- Not related to pharmacologic or toxic activity.

Spread of Oral Infections

INTRODUCTION

- Oral infection may start in the dental pulp and extend into the periapical and paraoral structures or may originate from the periodontal tissues and spread. Later on it outer cortical bone and various tissue spaces or discharge onto a free mucous membrane or skin surface. Mode of spread may be localized or extend diffusely route of spread of infection are lymphatic system, bloodstream or directly through the tissues. Mode of spread may depend on the type and virulence of the organisms, general health of the patient and anatomical life of the initial infection.
- The oral cavity is an unique structure exposed to the external environment. It harbors a vast array of microflora (350 species) that comprises of bacteria, fungi, mycoplasmas, protozoan and possibly viral flora. Most predominant microbes are bacteria.
- Unique thing about oral cavity is that it has number of sites with different environmental conditions. All these oral flora establish their own suitable microenvironments and live in complex mutually sustained communities. With aging, loss of teeth, the occurrence of caries and periodontal disorders, pregnancy or drug intake, the local environment of oral flora is disrupted. This is followed by conversion of normal flora to pathogenic flora.

These eventually may disseminate from the primary site of infection or foci of infection to result in systemic disorders.

- The foci of infection is defined as a primary localized or circumscribed area infected with microorganism which may or may not give rise to clinical manifestation.
- When these microorganisms or their toxins spread from foci of infection to distant sites via blood stream or lymphatics, these may eventually lead to some hypersensitive reactions or injury or secondary infection in a nearby or distant tissue or organs. It is called focal infections.
- An oral infection may originate in the dental pulp and extend through the root canals and into the periapical tissues or it may originate in the superficial periodontal ligaments and segmentally become dispersed through the spongy bone. Then it may perforate the outer cortical bone and spread in various tissue spaces or discharge onto a free mucous membrane or skin surface. It may become localized or extend diffusely.

CELLULITIS

- A painful swelling of the soft tissue of the mouth and face, sometime from a diffuse spread of purulent exudate along the facial planes that separate the muscle bundles.
- The term cellulitis is actually a misnomer because the process is not an inflammation of the cells but an acute condition in which purulent exudate, usually accompanied by virulent forms of bacteria, involves the fascial planes between the bundles of facial and perioral muscles. Cellulitis may occur from other causes in the head and neck area, but it is most commonly the result of extension of periapical abscess into the soft tissue. This occurs when the exudate erodes through the cortical plate of mandible or maxilla. When the erosion pathway reaches the gingival surface, it results in a small nodule that enlarges until it ruptures. The site of the opening of a sinus tract on the gingiva is commonly termed as **parulis**.
- Occasionally the exudate tracks onto the palate, producing a large tumor like mass. When a periapical abscess erodes into the maxillary sinus and destroy the intervening bone, the offending tooth is extracted.

- Communication between floor of sinus and oral cavity may result. Tract may remain permanently. If it becomes lined by epithelium from both sinus (antrum) and oral mucosal lining termed as oroantral fistula.
- Infections by microorganisms produce significant amount of hyaluronidase and fibrinolysis which act to breakdown or dissolve respectively hyaluronic acid, the universal intercellular cement substance and fibrin. Streptococci are particularly potent producers of hyaluronidase and are therefore causative organism. In case of cellulitis staphylococci (producing - hyaluronidase) are also pathogenic and frequently give rise to cellulitis.

Clinical Features

- Painful swelling of involved soft tissue, which is firm and brawny.
- Skin is inflamed, sometimes purplish.
- Pericoronitis (operculitis)
- Trismus
- Elevated temperature
- Leukocytosis
- Regional lymphadenitis
- In maxilla it perforates the outer cortical layer of bone above the buccinator attachment and causes swelling initially of upper half of face.
- In mandible, perforation of outer cortical plate below the buccinator, causes diffuse swelling of the lower half of the face which spreads cervically.
- If typical fascial cellulitis persists, the infection tends to become localized and facial abscess forms.

Histologic Features

- Diffuse exudation of polymorphonuclear leukocytes and occasional lymphocytes with considerable serous fluid and fibrin causing separation of connective tissue or muscle fibers.

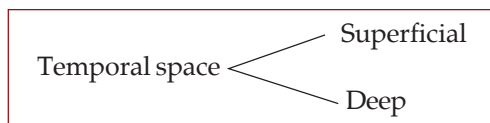
Treatment

- Administration of antibiotics and removal of cause.

INFECTIONS OF SPECIFIC TISSUE SPACES

Facial spaces of clinical significance are:

- Superficial compartment
 - Buccal space.
- Buccal vestibule
- Body of mandible
- Mental space
- Floor of the mouth
 - Sublingual space
 - Submandibular space
 - Submental space.
- Masticator space



- Submassetric space
- Superficial pterygoid space
- Parapharyngeal space
- Deep pterygoid space
- Parotid compartment
- Paratonsillar space.

INFRATEMPORAL SPACE

- It is bounded anteriorly by the maxillary tuberosity; posteriorly by the lateral pterygoid muscle, the condyle and temporal muscle, laterally by the tendon of the temporal muscle and the coronoid process, medially by lateral pterygoid plate and inferior belly of the lateral pterygoid muscle. The inferior portion of the infratemporal space is called pterygomandibular space and lies between the internal pterygoid muscle and the ramus of the mandible. Extending anteromedially from the infratemporal space and considered a part of it is the posterior zygomatic space. Infratemporal space contains the pterygoid plexus, the internal maxillary artery, the mandibular, mylohyoid lingual, buccinator and chorda tympani nerves and external pterygoid muscle.

Clinical Features

- Trismus and sometimes swelling of eyelids, especially if there is involvement of posterior zygomatic fossa.
- Involvement of pharynx may cause dysphagia and severe pain or feeling of pressure.
- Pterygomandibular space infection may arise through extension from a pericoronitis of a mandibular third molar and due to injection of local anesthetic solution into this space.
- Trismus and pain.
- Swelling of lateral posterior portion of soft palate.
- Injection of maxillary tuberosity with infected needle or solution cause infection of infratemporal fossa.

LATERAL PHARYNGEAL SPACE

- Bounded anteriorly by the buccopharyngeal aponeurosis, the parotid gland and pterygoid muscles.
- Posteriorly by the prevertebral fascia, laterally by carotid sheath and medially by the lateral wall of pharynx.
- Infection of this space with abscess formation impinge pharynx, causes difficulty in swallowing, breathing.
- Trismus occurs. Source of infection is third molar or second molar
- The lateral pharyngeal space communicates with mediastinum by prevertebral fascia, so that infection reaches this area.

RETROPHARYNGEAL SPACE

- It is bounded anteriorly by wall of pharynx, posteriorly by prevertebral fascia and laterally by lateral pharyngeal space and carotid sheath.
- Infection from medial extension in lateral pharyngeal space and an abscess may form displacing or pressing the buccopharyngeal fascia forward and impinging on the pharynx.
- As the prevertebral fascia extends inferiorly to the posterior mediastinum, it is possible for infection in the retropharyngeal space to spread down to the mediastinum.

PAROTID SPACE

- It contains the parotid gland and all invested associated structures, including facial nerve, auriculotemporal nerve posterior facial vein and external carotid, internal maxillary and superficial temporal artery.
- The gland is situated behind the ramus of the mandible and medially between the masseter and internal pterygoid muscle.
- Infection in parotid space reaching the gland from lateral pharyngeal space or by retrograde extension along the parotid duct, typically points medially or inferiorly and opens into the neck or oral cavity.
- Primary infection of parotid space breaks into the lateral pharyngeal space as the fascia is thin over the deep portion of the parotid space. Infection to temporal fossa may also occur.

SPACE OF THE BODY OF MANDIBLE

- It is enclosed by a layer of fascia derived from the outer layer of deep cervical fascia, which attaches to the inferior border of the mandible and then splits to enclose the body of the mandible. Superiorly it attaches to alveolar mucoperiosteum and muscles of facial expression which have this attachment on the mandible. The space contains the mandible anteriorly as well as covering periosteum, fascia, muscle attachments, blood vessels nerves, teeth and periodontal structures.
- Infections from incisor, cuspid or bicuspid teeth involves the space of the body of the mandible, there is induration or fluctuation of labial sulcus if the outer cortical plate is involved and if inner cortical plate is involved, the infection is restricted to floor of the mouth.
- Infection from molar teeth involving outer cortical plate results in swelling in the oral vestibule if the infection perforates the bone above the external oblique line, buccinator attachment. If perforation is below the line, the infection may point on the skin. Lingual spread from infected bicuspid or molar teeth is into the floor, when perforation of the bone is above the level of attachment of the mylohyoid muscle. Below mylohyoid infection extends to the sub maxillary space medially and posteriorly into the lateral pharyngeal space.

SUBMASSETRIC SPACE

- It is situated between masseter muscle and lateral surface of the mandibular ramus.
- Infection occurs from mandibular third molar, passing through the retromolar fossa and into the submassetric space. The patient may suffer from severe trismus, pain and facial swelling.

SUBMANDIBULAR

- Submaxillary, sublingual and submental are three spaces in submandibular region.
- *Submaxillary* - infection originates from mandibular molars and produces a swelling near the angle of jaw. It is most commonly involved to all facial and cervical tissue spaces. Involvement of submaxillary gland and nodes are seen due to close proximity. With sialadenitis and lymphadenitis it also involves submandibular spaces and extends to lateral pharyngeal space, carotid space, cranial fossa and mediastinum, pharynx and larynx may necessitate tracheostomy.

Sublingual Space

- Infection produces an obvious swelling in the floor of the mouth and may cause both dyspnea and dysphagia. Extension of the infection takes the same path as infection of submaxillary space.

Submental

- Infection in this area presents as an anterior swelling in the submental area. This may cause dyspnea and dysphagia. The spread of infection to that in the submandibular and sublingual spaces.

LUDWIG'S ANGINA

- It is a severe cellulitis beginning usually in the submaxillary space and secondarily involving the sublingual and submental space.
- Unless all the submandibular spaces are not involved it is not a Ludwig's angina.
- Chief source is mandibular molar, periapical or periodontal or

from penetrating injury of floor of mouth like gun shot or stab injury and osteomyelitis.

- Second and third molar be the most common source.
- When the infection perforates the base, it establishes drainage, it seeks the path of least resistance.
- Since outer cortical plate of mandible is thick in molar region, the lingual plate is most commonly perforated.

Clinical Features

- Swelling rapidly develops in floor of mouth and elevation of tongue is seen.
- Swelling is firm, painful and diffuse showing no evidence of localization.
- Difficulty in eating, swallowing and breathing.
- Fever, rapid pulse and fast respiration.
- Moderate leukocytosis.
- As swelling involves neck, infection spreads to parapharyngeal spaces and carotid sheath or pterygopalatine fossa.
- Cavernous sinus thrombosis with subsequent meningitis may be sequelae of spread of infection.

Lab Finding

- Mixed infection, but staphylococci mostly present.
- Fusiform bacilli in spiral forms, various staphylococci, diphtheroids have been cultured.

Treatment

- Antibiotics
- Edema of glottis necessitates tracheostomy to prevent suffocation.

CAVERNOUS SINUS THROMBOSIS

- Formation of thrombus in cavernous sinus or its communicating branches.
- Infections of head, face and intraoral structures above maxilla are prone to this disease.
- Infection of face and lip carried by facial and angular veins.
- Dental infection by pterygoid plexus.

Clinical Features

- Patients are extremely ill. Exophthalmous with edema of eyelids is seen.
- Paralysis of external ocular muscle, impairment of vision photophobia and lacrimation occurs.
- Headache, nausea, vomiting, pain, chills and fever are the common features.

Treatment

- Antibiotic has decreased mortality, but the condition is serious.

Benign and Malignant Tumors of Oral Cavity

Tumor

- The study of tumors of the oral and paraoral structures plays an important role in dentistry, because dentist is the major contributor in the diagnosis and treatment of these lesions. Although tumor constitute only a small minority of the pathologic conditions seen by the dentist, but they are having great significance since they have the potentiality to affect the health status and life of the patient. Dentist must be familiar with those lesions, so that when patient comes, we may either institute appropriate treatment or refer the patient to the proper therapist.
- It is simply a swelling of the tissue and the word does not imply a neoplastic process.

NEOPLASIA

(Willis 1952)

- A neoplasm can be defined as an abnormal mass of tissue the growth of which exceeds and is uncoordinated with that of the normal tissues and persists in the same excessive manner after cessation of the stimuli which evoked the change.

BENIGN NEOPLASMS OF THE EPITHELIAL TISSUE ORIGIN

PAPILLOMA

- Common benign neoplasm originating from surface epithelium. Frequently confused with fibroma.
- It is an exophytic growth made up of numerous, small finger like projections which result in a lesion with a roughened, verrucous or “CAULIFLOWER” surface.
- Well circumscribed pedunculated tumor, occasionally sessile.
- Found mostly on tongue, lips, buccal mucosa, gingiva and palate particularly that area adjacent to the uvula.
- Few mm in diameter but may vary up to cm.
- Occurs at any age even but mainly seen in young children.
- Common wart or verruca vulgaris is a frequent tumor of the skin analogous to the oral papilloma. It has been established that at least a considerable percentage of cases of the skin wart is caused by virus known as the papilloma virus.
- Actually multiple oral papillomas are quite uncommon except in certain situations such as the “Focal Dermal Hypoplasia Syndrome”. The usual lesion being a solitary papilloma.
- Oral papillomas of viral origin do exist in several other species of animals, however such as the dog and rabbit.
- Lesions that are histologically identical to the verruca vulgaris of the skin are frequently found on the lips and occasionally intraorally.
- Multiple hamartoma and neoplasia syndrome.
- Also called as Cowden’s syndrome. This is an autosomal dominant disease characterized by facial trichilemmomas associated with gastrointestinal tract, thyroid, central nervous system and musculoskeletal abnormalities as well as the oral lesions.
- Also considered as a cutaneous marker of breast cancer.

Histological Features

- Characterized by many long thin, finger like projections extending above the surface of the mucosa, each made up of a continuous layer of stratified squamous epithelium and containing a thin central connective tissue core which supports the nutrient blood vessels.

- Some exhibits hyperkeratosis due to local irritation.
- Essential feature is proliferation of the spinous cells in a papillary pattern.
- Presence of chronic inflammatory cells in connective tissue.

Treatment and Prognosis

- Excision including the base of the mucosa into which the stalk inserts.
- Removal should never be accomplished by an incision through the pedicle.
- Rare recurrence. Malignant degeneration is unlikely.
- Blue nevi are present at birth or appear in early childhood and persists unchanged throughout life.

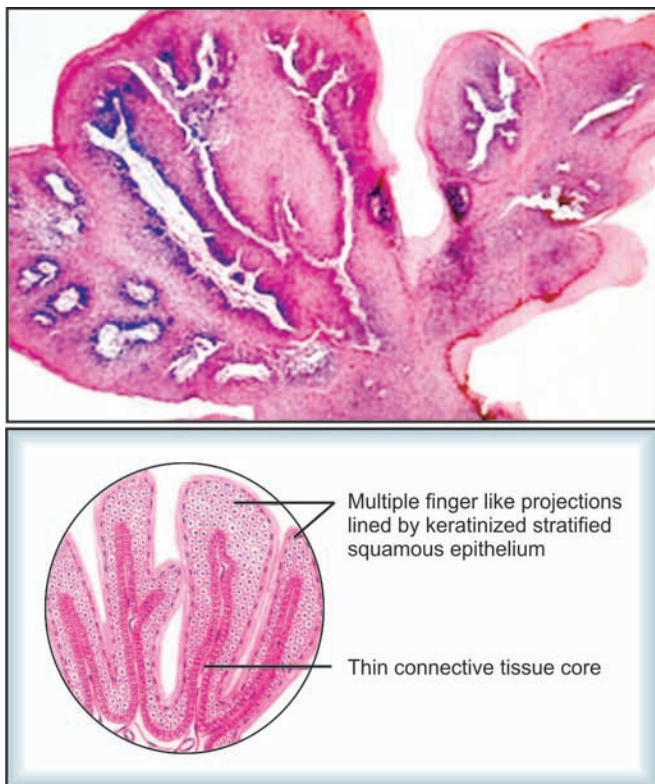


Fig. 12.1: Squamous papilloma

SQUAMOUS ACANTHOMA

- It's an uncommon lesion which probably represents a reactive phenomenon of epithelium, rather than a true neoplasm.
- It has not known relationship to the "clear cell acanthoma" which occurs with considerable frequency on the skin and may also be found on the lips.
- No distinct squamous acanthoma clinical features are seen by which it can be suspected.
- May occur at any site on the oral mucosa.
- Usually seen in older adults.
- Characterized by small, flat or elevated, white, sessile or pedunculated lesion of the mucosa.
- Lesion is histologically distinctive and consists of a well-demarcated elevated and umbilicated epithelial proliferation with a markedly thickened layer of orthokeratin and underlying spinous layer of cells.
- Caused by trauma and developed its characteristic morphology through series of epithelial alterations beginning with a localized pseudoepitheliomatous hyperplasia.

Treatment

- Excision and recurrence is rare.

KERATOACANTHOMA

- It is a lesion which clinically and histologically resembles epidermoid carcinoma.
- Also called self-healing carcinoma, molluscum sebaceum, molluscum pseudocarcinomatous verrucoma.
- It's a benign lesion.

Etiology

- Both genetic and viral factors and sometime chemical carcinogens.

Clinical Features

- Male : Female ratio is 2:1.
- Seen in age of 50-70 years.

- Commonly seen in lips (8.1%). Intraoral lesion is quite uncommon. Approximately equally distributed between upper and lower lips.
- Appears as an elevated umbilicated or crateriform one with depressed central core or plug.
- Seldom over 1.0-1.5 cm in diameter.
- Often painful and regional lymphadenopathy present.
- It begins as a small, firm nodule that develops to full size over a period of 4-8 weeks, persists for 4-8 weeks then undergoes spontaneous regression over next 6-8 week by expulsion of the keratin core resorption of the mass.
- Recurrence is rare.

Histologic Features

- Consists of hyperplastic squamous epithelium growing into the underlying connective tissue.
- Surface is covered by a thickened layer of parakeratin or orthokeratin with central plugging.
- Epithelial cells don't usually show atypia but the deep leading margin of the tumor, islands of epithelium often appear to be invading and frequently. This area can not be differentiated from epidermoid carcinoma.
- Chronic inflammatory cells seen in connective tissue.
- Striking feature is, adjacent epithelium is elevated toward the central portion of the crater, and then an abrupt change in the normal epithelium occurs as the hyperplastic acanthotic epithelium is reached.
- Adjacent epithelium should be included in the biopsy to differentiate between keratoacanthoma and epidermoid carcinoma.

Treatment

- Surgical excision.
- Spontaneous regression does not occur in every case.

PIGMENTED CELLULAR NEVUS

- Also called pigmented mole and benign melanocytic nevus.
- A nevus is defined as a congenital, developmental tumor like malformation of the skin or mucous membrane.

- It's a skin lesion containing melanin pigments. Pigmented nevus is a superficial lesion composed of so-called nevus cells, hence the term "cellular" nevus.
- It is seen occasionally in the oral cavity but occurs far more frequently on the skin.

CLASSIFICATION

(A) Congenital

- Small
- Garment

(B) Acquired

- Intradermal nevus (common mole)
- Junctional nevus
- Compound nevus
- Spindle cell/epitheloid cell nevus
- Blue nevus (Jadassohn-Tieche)

Clinical Features

- Small nevi are more than 1cm in diameter and usually 3-5 cm and garment nevi are more than 10 cm and can cover large areas of the skin.
- Congenital nevi occur in 1-2.5 percent of neonates and may change from flat, pale tan macules to elevated, verrucous, hairy lesions.
- Approximately 15 percent seen in head and neck. Intraoral occurrence is rare.
- Acquired nevi are extremely common. Appear about the 8th month of life and increase in number with aged reach their peak at third decade of life.
- Number of nevi is genetically determined. Interestingly the number of nevi begin to decrease as one ages, so that elderly persons average fewer nevi than young adults.
- Intradermal nevus is one of the most common lesions of the skin. Most often dozens, scattered over the body.
- Common mole may be smooth flat lesion or may be elevated above the surface. It may or may not exhibit brown pigmentation and it often shows strands of hair growing from its surface.
- This type of mole seldom occurs on the soles of the feet, the palms of hands or the genitalia.
- Junctional nevus may appear clinically similar to intradermal nevus. The distinction being chiefly histologic.

- Compound nevus is a lesion composed of two elements an intradermal nevus and overlying junctional nevus.
- Spindle cell and epitheloid cell nevus occurs chiefly in children only 15 percent in adults.
- May appear histologically similar to malignant melanoma in adult.
- Seldom, however does this lesion exhibit clinically malignant features.

***Essentially this Lesion is Clinically Benign
but Histologically Malignant***

- Blue nevi are a true mesodermal structure composed of dermal melanocytes which only rarely undergo malignant transformation.
- Occurs chiefly on buttocks, on the dorsum of the feet and hands, on the face and occasionally on other areas.
- The lesion is smooth, exhibits hair growing from its surface and varies in color from brown to blue or bluish black.

Oral Manifestations

- Acquired nevi except spindle cell or epitheloid cell nevus occasionally occur in the oral mucosa.
- Blue nevi are also called as intraoral nevi.
- Intraoral nevi occur in all decades except the first and M:F ratio is 1:2.
- Site → Buccal mucosa, hard palate, lip and gingiva.

Histologic Features

- Theories of origin of the nevus cell is controversial.
- Nevus cells are derived from neural crest cell.
- Nevus cells are large discrete cells each with an avoid, vesicular nucleus and pale cytoplasm.
- Tend to be grouped in sheets or cords and may contain granules of melanin pigments in their cytoplasm.
- The arrangement of these cells in an alveolar pattern is referred to as theques.
- Multinucleated giant cells are sometimes seen but one of little prognostic significance.

- In the intradermal nevus, the nevus cells are situated within the connective tissue and are separated from overlying epithelium by a well defined band of connective tissue. The nevus cells are not in contact with the surface epithelium.
- In the junctional nevus, this zone of demarcation is absent and the nevus cells contact and seem to blend into the surface epithelium. This overlying epithelium is usually thin and irregular and shows cells apparently crossing the junction and growing down into the connective tissue so called “ABTROPFUNG” or “DROPPING OFF” effect. This junctional activity has serious implications because junctional nevi have

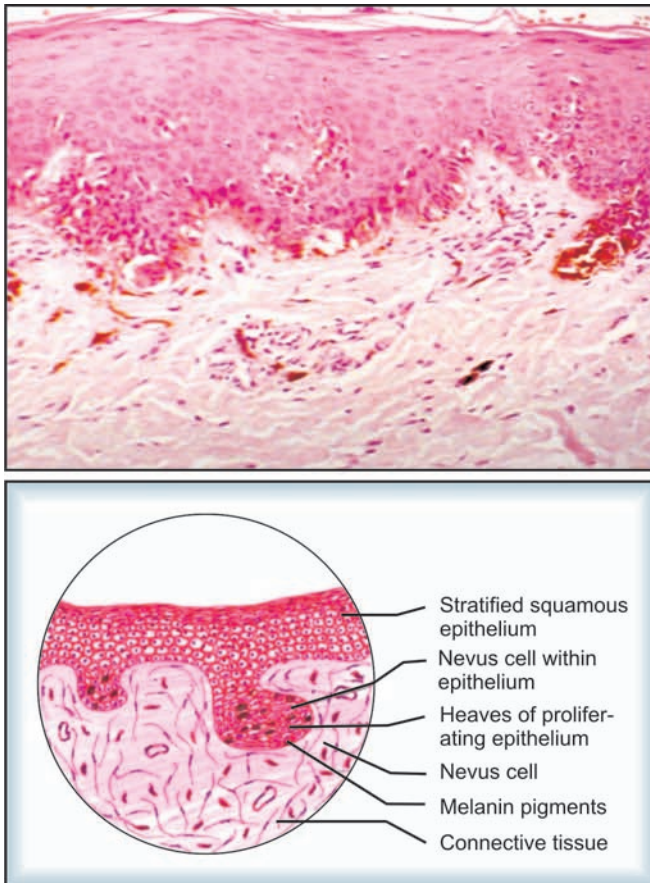


Fig. 12.2: Junctional nevus

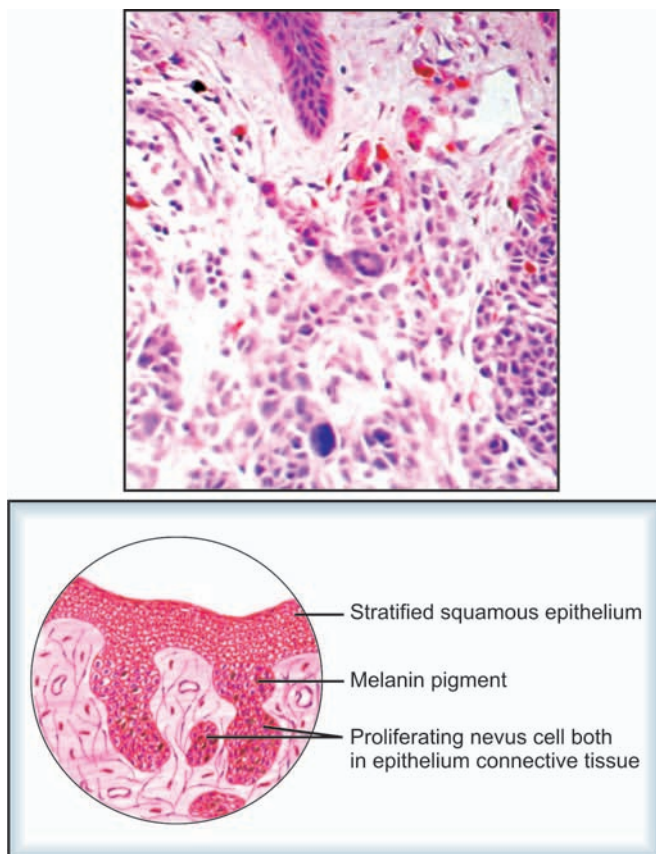


Fig. 12.3: Compound nevus

been known to undergo transformation into malignant melanomas.

- The compound nevus shows features of both compound and junctional nevus. Nests of nevus cells are dropping off from the epidermis, while large nests of nevus cells are also present in the dermis.
- The spindle cell nevus is commonly composed of pleomorphic cells of three basic types
 - Spindle cells
 - Oval or epitheloid cells
 - Mononucleated and multinucleated cells.

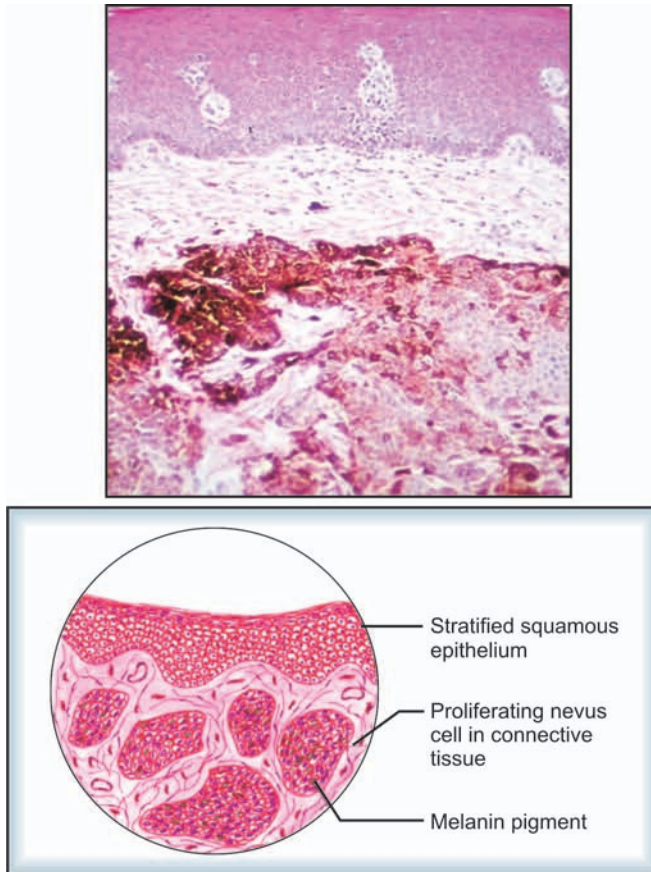


Fig. 12.4: Intradermal nevus

- Arranged in well circumscribed sheets and there is generally considerable junctional activity.
- Blue nevus is of two types
 - Common blue
 - Cellular blue
- Common blue nevus elongated melanocytes with long branching dendritic processes lie in bundles usually oriented parallel to the epidermis in the middle and lower 1/3rd of the dermis.
- There is no junctional activity.
- In the cellular blue nevus an additional cell type is present. A large, round or spindle cell with a pale vacuolated cytoplasm.
- These cells commonly are arranged in an alveolar pattern.

Treatment and Prognosis

- Excision if causing irritation or change in size, deepen in colour and become ulcerated.
- Trauma to an intradermal nevus will not induce malignancy.
- On the other hand it is now well recognized that the congenital nevus have a great risk for transformation to malignant melanoma.
- Surgical removal of all intraoral pigmented nevi is recommended as a prophylactic measure because of the constant chronic irritation of the mucosa in nearly all intraoral sites occasioned by eating, teeth brushing.

“PREMALIGNANT” LESIONS OF EPITHELIAL TISSUE ORIGIN

Premalignant Lesion

- A benign morphologically altered tissue, that has a greater than normal risk of malignant transformation.

Premalignant Condition

- A disease (or) a patient habit that does not necessarily alter the clinical appearance of local tissue but always associated with a greater than normal risk of precancerous (or) cancer development in that tissue.

LEUKOPLAKIA

- It is a term that has been used for many years to indicate a white patch or plaque occurring on the surface of a mucous membrane, not only that of the oral cavity, but also that of the vulva, uterine cervix, urinary bladder, renal pelvis and upper respiratory tract.
- In some instance the term has been used to describe a white patch on the mucosa which will not rub or strip off.
- At the opposite extreme, the diagnosis of leukoplakia has been based on strict histologic criteria and such a diagnosis has often been made even though clinically the lesion does not appear as a white patch.

Histologic Terminology

- Keratosis
- Hyperkeratosis simplex

- Leukokeratosis
- Hyperkeratosis complex
- Hyperkeratosis
- Non-specific Focal Keratosis
- Pachyderma oralis
- Leukoplakia
- Interepithelial carcinoma
- The term leukoplakia are used here will be simply only the clinical feature of a white plaque on the mucosa.

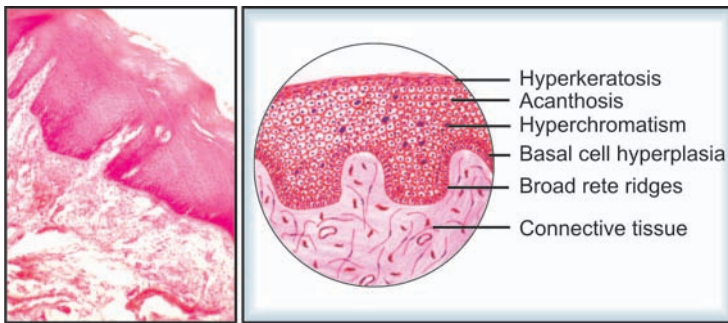


Fig. 12.5: Leukoplakia

Etiology

- Tobacco
- Alcohol
- Oral sepsis
- Local irritation
- Syphilis
- Vitamin deficiency
- Endocrine disturbance
- Galvanism
- Actinic radiation.

Clinical Features

- Lesions of oral leukoplakia show considerable variation in size, location and clinical appearance.
- More common in men than in women and it is seen chiefly in older age group.

Site

- Buccal mucosa and commissures
- Alveolar mucosa
- Tongue
- Lip
- Hard and soft palate
- Floor of the mouth
- Gingiva
- Extent of involvement may vary from small, well localized, irregular patches to diffuse lesions covering a considerable portions of the oral mucosa.
- On clinical examination, patches of leukoplakia may vary from a nonpalpable, faintly translucent white area to thick, fissured, papillomatous, indurated lesion.
- The surface of the lesion is often finely wrinkled or shriveled in appearance and may feel rough on palpation.
- Lesions are white, gray or yellowish-white but with heavy use of tobacco may assume a brownish yellow color.
- Sharp described three stages of leukoplakia.
- The earliest lesion being a non-palpable, faintly translucent, white discoloration.
- Later localized or diffuse, slightly elevated plaques of irregular outline develop. These are opaque white and may have a fine granular texture.
- In some instances the lesions progress to thickened, white lesions, showing induration fissuring and ulcer formation.
- Little doubt that a certain percentage of cases of leukoplakia will undergo transformation into epidermoid carcinoma.

Histologic Features

Hyperorthokeratosis

- Moderate amount of orthokeratin present on the surface of normal oral epithelium.
- Hyperkeratosis refers to an abnormal increase in thickness of this orthokeratin layer or stratum corneum in a particular location.
- Although it is some what seen in normal oral mucosa but for some sites it is abnormal.

Hyperparakeratosis

- Parakeratin differs from orthokeratin in the persistence of nuclei or nuclear remnants in the keratin layer.
- Presence of parakeratin where it should not be found is abnormal.
- The stratum granulosum is often thickened and extremely prominent in cases of hyperorthokeratosis but is seldom seen even in severe cases of hyperparakeratosis.

Acanthosis

- Thickness of the spinous layer or prickly cell also varies considerably between different areas of the oral cavity.
- Acanthosis refers to the abnormal thickening of the spinous layer for a particular location.

Dysplasia

Criteria for epithelial dysplasia are:

- Red and particularly abnormal mitosis.
- Individual cell keratinization.
- Epithelial pearls within the spinous layer.
- Alterations in the nuclear - cytoplasmic ratio.
- Loss of polarity and disorientation of cells.
- Hyperchromatism of cells.
- Large prominent nucleoli.
- Dyskeratosis or nuclear atypism including giant nuclei.
- Poikilokaryosis or division of nuclei without division of cytoplasm.
- Basilar hyperplasia.
- Carcinoma in situ or intraepithelial carcinoma.
- A definite distinction can not always be drawn between dysplasia and CIS (Carcinoma in Situ).
- When exhibiting top to bottom change particularly with respect to basilar hyperplasia must undoubtedly be diagnosed as carcinoma in situ, provided of course that it has not progressed to that point of true invasion of the connective tissue.

MALIGNANT POTENTIAL

- Percentage of cases of leukoplakia which undergo malignant transformation, despite the marked variation in follow-up periods.
- It ranges from 1.4 - 6.0%, averaging slightly over 4.0%.

Treatment

- Elimination of any recognizable irritating factors.
- Administration of vitamin 'A', B-complex and estrogens.
- X-ray therapy.
- Fulguration.
- Surgical excision.
- Topical chemotherapy.
- Discontinuation of tobacco or alcohol.
- Correction of any possible malocclusion.
- Replacement of ill-fitting dentures.
- Relatively small lesions may be totally excised or cauterized.
- Field cancerization done.
- Extensive lesions treated by multiple stage stripping procedure with or without skin grafting.

Differential Diagnosis

- Lichen planus
- Chemical burns
- Syphilitic mucous patches
- Mycotic infections (candidosis)
- Psoriasis
- Lupus erythematosus
- White spongy nevus
- White folded gingivostomatitis

LEUKOEDEMA

- It is an abnormality of the buccal mucosa which clinically resembles early leukoplakia but appears to differ from it in certain respect.

Clinical Features

- Gross appearance of leukoedema varies a filmy opalescence of the mucosa in the early stages to a more definite greyish-white cast with a coarsely wrinkled surface in the later stages.
- Occur bilaterally in the majority of cases and frequently involve most of the buccal mucosa, extending onto the oral surface of the lips.
- Mostly noticeable along the occlusal line in the bicuspid and molar region. In some cases desquamation occurs, leaving the surface eroded.

Etiology

- Unknown
- No correlation with tobacco.

Histologic Features

- Increase in thickness of the epithelium.
- Intracellular edema of the spinous or malpighian layer → A superficial parakeratotic layer, several cells in thickness and broad rete pegs which appear irregularly elongated.
- Characteristic edematous cells appear extremely large and pale and they present reticular pattern.
- Cytoplasm appears lost and nuclei appear absent, clear or pyknotic.

Clinical Significance

- This is a lesion in which leukoplakia is more apt to develop than in normal epithelium.
- Not associated with malignancy or potential malignant alteration.
- Since leukoedema appears to be simply a variant of normal mucosa, no treatment is necessary.

INTRAEPITHELIAL CARCINOMA

- Also called as carcinoma in situ.
- Arises frequently on the skin but also occurs on mucous membranes, including oral cavity.

- It represents a precancerous dyskeratotic process.
- It is a laterally spreading, intraepithelial type of superficial epithelioma or carcinoma.
- In this stage it does not exhibit invasive malignant properties since metastasis can not occur without infiltration of tumor cells into connective tissue and consequently accessibility to lymphatic or blood vessels, metastasis is impossible in intra-epithelial carcinoma
- Bowen's disease is a special form of intra-epithelial carcinoma occurring with some frequency on skin, particularly in patients who have had arsenical therapy often associated with the development of internal or extracutaneous cancer.

Clinical Features

- Clinical appearance was that of a leukoplakia in 45 percent of the lesion, an erythroplakia in 16 percent of lesion and 9 percent in combination. White and ulcerated lesion in 5 percent and red and ulcerated lesion in 1percent .
- Lesions reported to occur at all intraoral site but most common is floor of the mouth, tongue, lips in male
- Male : Female is 1.8:1 (Tend to occur in elderly person).

Histologic Features

- Remarkable range of variation in histologic appearance.
- Keratin may or may not be found on the surface of the lesion but if present is more apt to be parakeratin than orthokeratin.
- Appears to be hyperplasia of the altered epithelium, while others there is atrophy.
- An increased nuclear/cytoplasmic ratio and nuclear hyperchromatism are sometimes seen.
- All the above changes seen only in surface epithelium, confined to basement membrane.

Treatment and Prognosis

- No uniformly accepted treatment.
- Lesion is surgically excised, cauterized and even exposed to solid CO₂.
- Spontaneous regression also seen in uterine cervix.

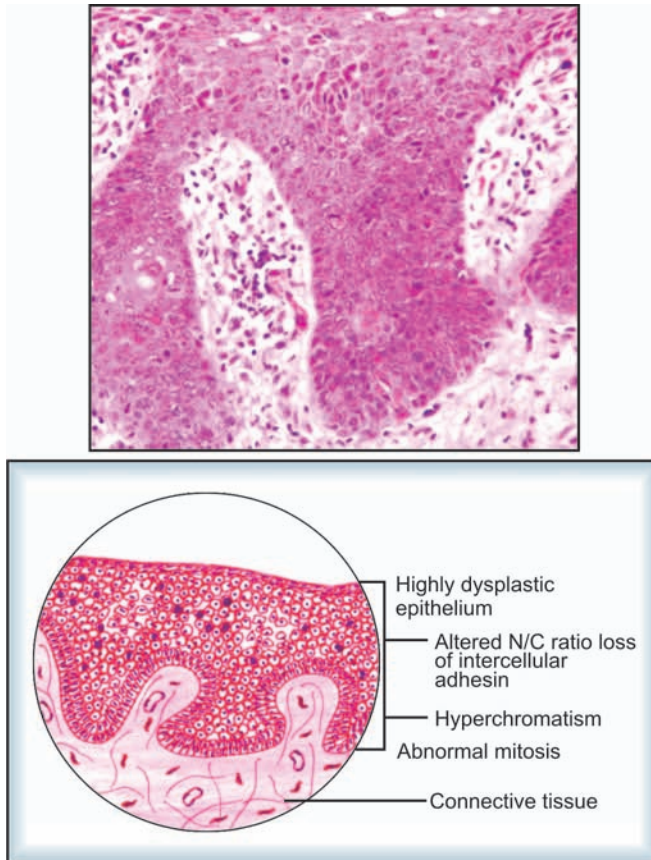


Fig. 12.6: Carcinoma in situ

ERYTHROPLAKIA

- Also called as erythroplasia of Queyrat.
- Disease originally described under the name “erythroplasia by Queyrat” in 1911 as a lesion occurring on the glans penis of an elderly syphilitic person.
- Also seen in vulva and oral mucosa since then investigators recognize that erythroplakia is a clinical entity and represent a lesion of mucous membrane which histologically in a large percentage of cases, exhibits epithelial changes ranging from mild dysplasia to carcinoma in situ and even invasive carcinoma.

Similar Lesions

- Candidosis
- Tuberculosis
- Histoplasmosis
- Denture irritation.

Clinical Features

- Appear to be three different clinical manifestation of erythroplakia in the oral cavity.
- Homogeneous form which appears as a bright red, soft velvety lesion with straight or scalloped well demarcated margins, often quite extensive in size, commonly found on the buccal mucosa and sometimes on the tongue and floor of the mouth.
- Erythroplakia interspersed with patches of leukoplakia in which the erythematous areas are irregular and often not as bright red as the homogeneous form, most frequently seen on the tongue and floor of the mouth.
- Soft, red lesions that are slightly elevated with an irregular outline and a granular or finely nodular surface speckled with tiny white plaques often referred to as "SPECKLED LEUKOPLAKIA" or more properly "SPECKLED ERYTHROPLAKIA".
- Oral erythroplakia is an uncommon disease.
- It does not have any sex specification and it occurs most frequently in 6th to 7th decades of life.

Site

- Floor of the mouth, retromolar area followed by tongue, palate and mandibular mucosa and sulcus.

Histologic Features

- Erythroplakia are histologically either invasive epidermoid carcinoma, C.I.S or epithelial dysplasia at the time of biopsy.
- The region for the red appearance of the lesion clinically becomes apparent when these lesions are studied histologically, it will be found that the connective tissue retepegs extend very high into the epithelium and that the epithelium over the tips of these retepegs is often very thin. Capillaries are dilated. Absence of ortho and parakeratin layer.

Treatment

- Application of 5 - fluorouracil in the oral cavity is experimental.

ORAL SUBMUCOUS FIBROSIS (OSMF)

- OSMF is a peculiar disease which is considered to be a precancerous condition.
- Occurs chiefly in South East Asia. Occasionally seen in U.S.A.
- *Definition:* It is an insidious chronic disease affecting any part of the oral cavity and sometimes the pharynx. Although occasionally preceded by and or associated with juxta-epithelial inflammatory reaction followed by a fibroelastic change of the lamina propria with essential atrophy leading to stiffness of the oral mucosa and causing trismus and inability to eat.

Etiology

- Peculiar dietary component like chilies, strongly irritating spice commonly used in India.
- Vit 'B' deficiency.
- Protein
- Betel nut chewing.

Clinical Features

- Characterized by a burning sensation of the mouth, particularly when eating spicy foods.
- This is accompanied or followed by the formation of vesicles (on palate), ulcerations or recurrent stomatitis with excessive salivation or xerostomia and defective gustatory sensation.
- Patient develops stiffening of certain areas of oral mucosa with difficulty in opening the mouth and swallowing, thus stimulating systemic sclerosis or "scleroderma".
- Mucosa eventually becomes blanched and opaque (although erythroplakic areas are some time seen) and fibrotic bands appear usually involving the buccal mucosa, soft palate lips and tongue.
- Most common in between 20-40 years of age but can occur at any decades of life.

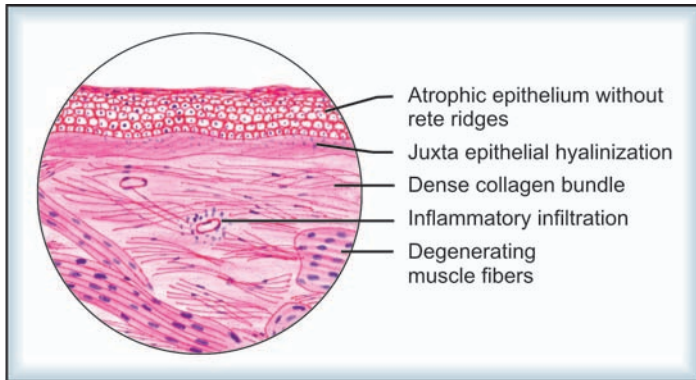


Fig. 12.7: Oral submucous fibrosis

Histologic Features

- Oral epithelium is almost invariably extremely atrophic with complete loss of rete pegs.
- Epithelial atypia may also be present.
- Underlying connective tissue shows severe hyalinization with homogenization of collagen bundles.
- Fibroblasts are markedly diminished in number and the blood vessels are completely obliterated or narrowed.
- Chronic inflammatory cells seen.
- Basal cell is a pluripotential cell which may develop in hair, sebaceous glands.

Treatment and Prognosis

- Systemic corticosteroids and local hydrocortisone have appeared to offer some temporary remissions.

MALIGNANT TUMORS OF EPITHELIAL TISSUE ORIGIN

BASAL CELL CARCINOMA

- Also called as basal cell epithelioma or rodent ulcer.
- It develops most frequently on the exposed surfaces of the skin, face and the scalp in middle aged or elderly persons.

- Most common type of carcinoma in men.
- Exhibits practically no tendency for metastasis and for this reason has often called a benign carcinoma.
- Head and neck is the site of the primary tumor in 85 percent of the cases.

Etiology

- There is a relation between prolonged exposure to sunlight and skin cancer.
- It is due to UV radiation, ionizing radiation, burns scars as well as other scars and many different types of chemicals.
- Sometimes general atrophy may develop skin cancer.

Clinical Features

- Age 40 years or later.
- Common in men than women.
- Usually begins as a small, slightly elevated papule which ulcerates, heals over and then breaks down again.
- Eventually the crusting ulcer, which appears superficial, develops a smooth, rolled border representing tumor cells spreading laterally beneath the skin.
- Untreated lesions continue to large, infiltrate adjacent and deeper tissues and may even erode deeply into cartilage or bone.
- Most frequently seen in middle 1/3rd of the face but may occur anywhere in the skin.

Histologic Features

- Characterized by the appearance of nests, islands or sheets of cells showing indistinct cell membranes with large, deeply staining nuclei and variable number of mitotic figures.
- Periphery of the cell nests is composed of a layer of cells, usually well polarized, that are suggestive of cells of the basal layer of skin. Sweat glands or squamous epithelium and eventually keratin.

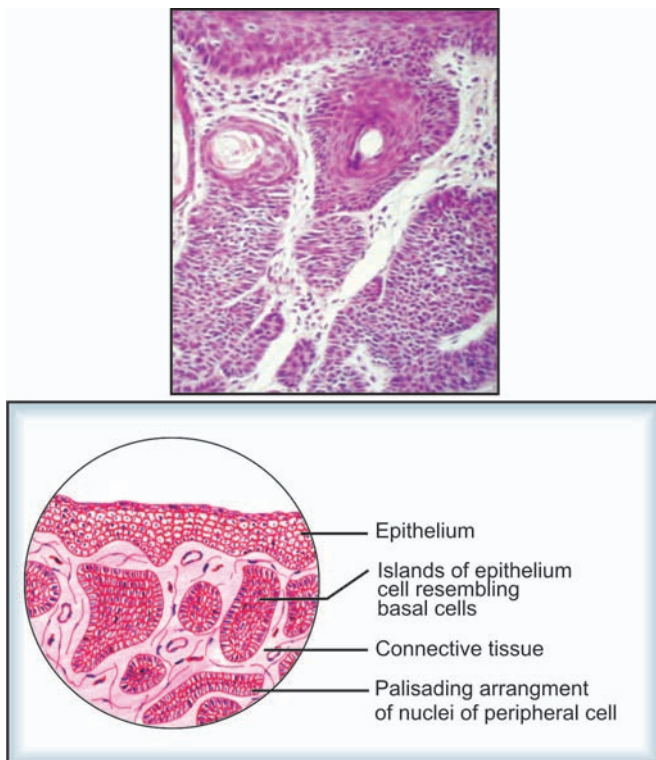


Fig. 12.8: Basal cell carcinoma

Types

- Adenoid B.C.C.
- Cystic B.C.C.
- Keratotic B.C.C.
- Primordial B.C.C.

Treatment and Prognosis

- Surgical excision and X-ray radiation.
- Good prognosis.
- Field cancerization.

EPITHELIAL DYSPLASIA

- It is defined as a premalignant change in epithelium characterized by a combination of cellular and architectural alterations.
- Dysplasia means disordered cellular development.
- Epithelial dysplasia is characterized by cellular proliferation and cytologic changes.
- These include [Adapted from Kramer, et al. 1978]
 - Loss of polarity of the basal cells.
 - Presence of more than one layer of cells having basaloid appearance.
 - Increased nuclear cytoplasmic ratio.
 - Drop shaped rete pegs.
 - Irregular epithelial stratification.

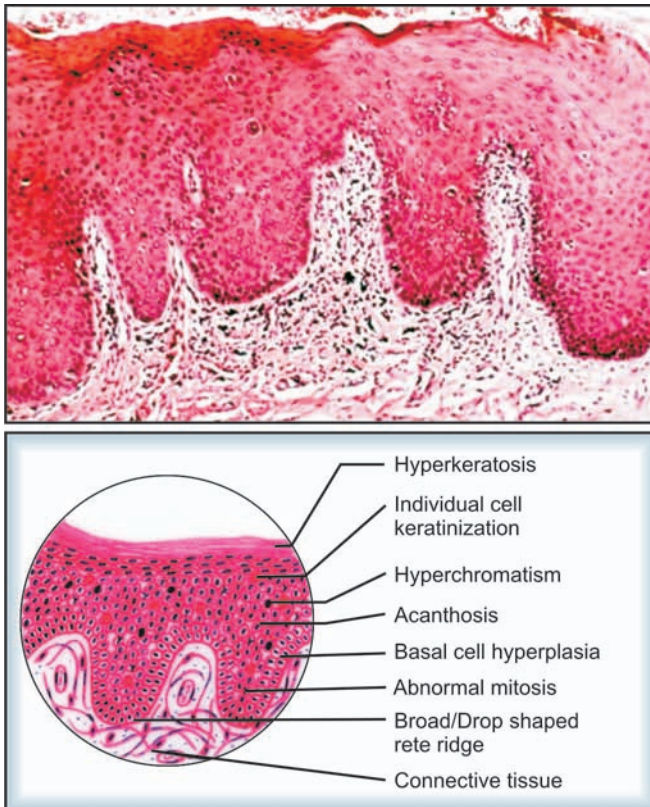


Fig. 12.9: Epithelial dysplasia

- Increased number of mitotic figures.
- Presence of mitotic figures in the superficial half of the epithelium.
- Cellular pleomorphism.
- Nuclear hyperchromatism.
- Enlarged nucleoli.
- Reduction of cellular cohesion.
- Keratinization of single cells or cells groups in the prickle layer.
- Epithelial dysplasia is usually divided into three categories (according to microscopic features)
 - Mild
 - Moderate
 - Severe.
- The rate at which epithelial dysplasia will progress from it's mildest to its most severe forms will vary considerably among individuals and may range from months to years.
- Some mild and incipient forms of epithelial dysplasia will regress (reverse) and the epithelium will revert to normal when the inciting factor is removed. In some cases reversal may not be possible, even after the removal of the causative factor. In some cases, removal may slow the rate of progression.
- Moderate and severe form of epithelial dysplasia may or may not regress by simply removing the causative factor.
- Areas of epithelial dysplasia often exhibit a chronic lymphocytic infiltrate in the adjacent connective tissue, and lymphocytes extend into the deeper layers of the dysplastic epithelium.

SQUAMOUS CELL CARCINOMA

- Also called as epidermoid carcinoma.
- Most common malignant neoplasm of the oral cavity.

Etiology

- Tobacco
- Alcohol
- Syphilis
- Nutritional deficiency and dentures
- Sunlight (Lip cancer)
- Heat (Heat from pipe stem in lip cancer)

- Trauma
- Sepsis
- Virus.

Histologic Features

- In general they tend to be moderately well differentiated neoplasms with some evidence of keratinization.
- Highly anaplastic areas seen but uncommon.
- Well differentiated epidermoid carcinoma consists of sheets and nests of cells with obvious origin from squamous epithelium.
- These cells are generally large and show a distinct cell membrane although intercellular bridges or tonofibrils often can not be demonstrated.
- Nuclei of the cell are large hyperchromatic.
- Mitotic figures are typical presence of individual cell keratinization and the formation of numerous epithelial or keratin, pearls of varying size.
- In a typical lesion epithelial pearls are seen invading into the connective tissue.
- Moderately differentiated epidermoid carcinoma shows altered growth rate of individual cell which is more rapid.
- Greater number of mitotic figures with greater variation in size, shape and tinctorial reaction of a differentiated squamous cell, i.e. the formation of keratin.
- Poorly differentiated carcinomas bear little resemblance to their cell of origin and often will present diagnostic difficulty because of the primitive and uncharacteristic histologic appearance of the malignant rapidly dividing cells.
- These cells show an even greater lack of cohesiveness and are extremely various.

Grade (Border)

- **Grade I:** Highly differentiated, i.e. cells were producing much keratin.
- **Grade II:** Very poorly differentiated, i.e. cells were highly anaplastic and showed practically no keratin formation.

Treatment

- No specific treatment. It depends on the grading and staging of the carcinoma.



Fig. 12.10: Squamous cell carcinoma

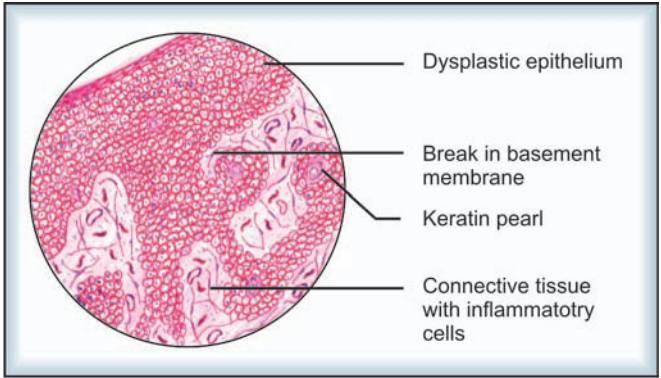


Fig. 12.11: Moderately differentiated carcinoma

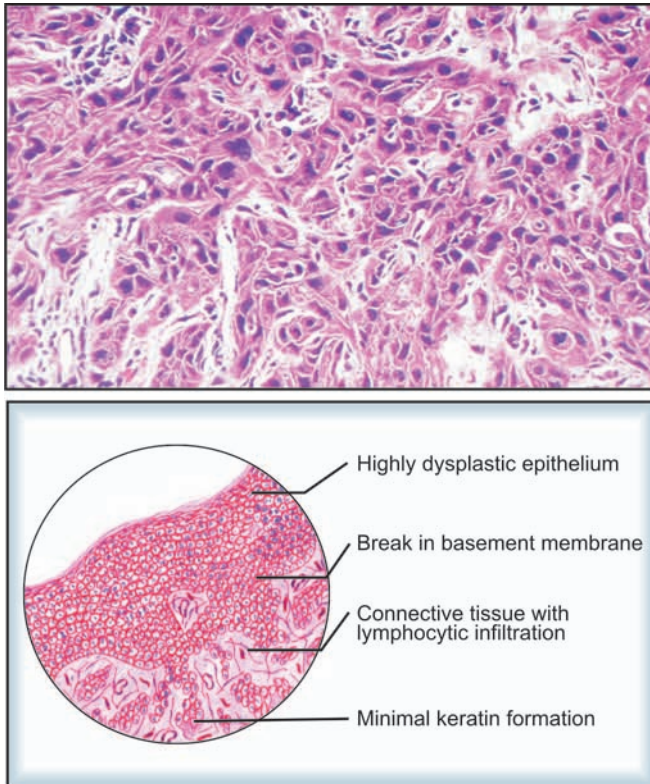


Fig. 12.12: Poorly differentiated carcinoma

VERRUCOUS CARCINOMA

- It is form of epidermoid carcinoma of the oral cavity which is mainly seen in oral cavity and also in larynx, esophagus, nasal fossae and paranasal sinuses, external auditory meatus, lacrimal duct, skin, scrotum, penis, vulva, vagina, uterine cervix, perineum, leg and odontogenic cyst lining.
- Oral lesions are slowly growing, chiefly exophytic and only superficially invasive at least until late in the course of the disease and has a low metastatic potential.
- Site → Head and neck.

Clinical Features

- Age → Elderly patient-> 60-70 years
- Sex → Male 75% Female 25%
- Site → Buccal mucosa. Gingival or alveolar ridge, palate and floor of the mouth.
- Neoplasm is chiefly exophytic and appears papillary in nature with a pebbly surface which is covered by a white leukoplakic film.
- Lesions commonly have rugae like fold and deep clefts on them.
- Regional lymph nodes are tender and enlarged, simulating metastatic tumor but this node involvement is usually inflammatory.
- Pain and difficulty is common complaint but bleeding is rare.

Etiology

- High percentage in tobacco chewing.
- Low percentage is snuff or heavy smoking.

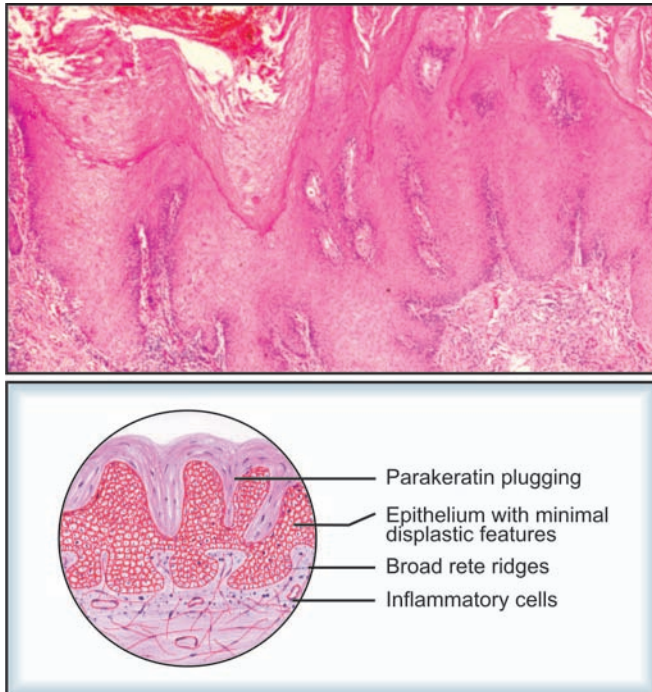


Fig. 12.13: Verrucous carcinoma

Histologic Features

- Generally, marked epithelial proliferation with down growth of epithelium into the connective tissue but usually without a pattern of true invasion.
- Epithelium is well differentiated and shows little mitotic activity, pleomorphism or hyperchromatism.
- Parakeratin plugging is the hallmark of verrucous carcinoma.
- Significant chronic inflammatory cell infiltration in the underlying connective tissue.

Treatment and Prognosis

- Surgery, X-ray radiation or a combination of both.
- Prognosis is better.

SPINDLE CELL CARCINOMA

- Also called carcinosarcoma, pseudosarcoma, polypoid squamous cell carcinoma and lane tumor.
- This is an interesting, unusual and controversial tumor which occur chiefly in the upper respiratory tracts.
- Now majority of people believe that, it is epithelial and the tumor is a variant of squamous cell carcinoma.

Clinical Features

- Site -> Greatest frequently on the lower lip, tongue, alveolar ridge or gingiva.
- Characterized by swelling, pain and the presence of a non healing ulcer.
- Initial lesion appeared either with polypoid, exophytic or endophytic configuration.
- May be seen in previously exposed therapeutic radiation in a tissue interval of 1.5-10 years with a mean of 7 years.

Histologic Features

- It is bimorphic or biphasic tumor which although almost always ulcerated but show foci of surface epidermoid carcinoma or epithelium dysplasia of surface mucosa, usually just at the periphery and often quite limited.

- Proliferation and dropping off of basal cells to spindle cell elements is a common phenomenon.
- The bulk of the tumor may be either fasciculated, myxomatous or streaming.
- In fasciculated form, the cells are elongated with epithelial nuclei, although pleomorphic cells are also common number of mitosis may vary.
- Giant cells, both benign appearing of the foreign body type and the bizarre, pleomorphic, a typical cell may be found.
- Osteoid formation within the tumor component is sometimes seen microscopic invasion of subjacent structures is evident.

Treatment and Prognosis

- Surgical removal of the tumor with or without radicle neck dissection alone or in combination with radiation is the treatment.
- If metastasis is there, then, poor prognosis.

ADENOID SQUAMOUS CELL CARCINOMA

- Also called adenoacanthoma and pseudoglandular squamous cell carcinoma.
- It is an interesting tumor of the skin which also occurs with considerable frequency on the lips.
- There is a considerable evidence that the tumor originates from the pilosebaceous structures and also from senile keratosis with acantholysis.

Clinical Features

- Age → Early age → 20 years.
- Sex → Common in female than male.
- Lesions of the skin appear as simply elevated nodules that may show crusting, scaling or ulceration. Sometimes elevated or rolled border is seen.

Histologic Features

- There is proliferation of surface dysplastic epithelium into the connective tissue as in the typical epidermoid carcinoma.

- The lateral or deep extensions of this epithelium show the characteristic solid and tubular ductal structures which typify the lesion.
- These duct like structures are lined by a layer of cuboidal cells and often contain or enclose acantholytic or dyskeratotic cells.

Treatment and Prognosis

- Generally treated by surgical excision.
- Rarely metastasize and cause death of patients.
- Recurrence is common.

LYMPHOEPITHELIOMA

- Also called as transitional cell carcinoma.
- This is an unusual group of malignant neoplasm exhibiting features in nasopharynx, oropharynx, tongue, tonsil and anatomical associated structures such as nasal chamber and paranasal sinuses.
- Arise from mucosa of these areas.
- This group of neoplasm consists of lymphoepithelioma, transitional cell carcinoma and undifferentiated squamous cell carcinoma.
- Lymphoepithelioma occurs chiefly in the nasopharynx of young or middle-aged persons often manifested due to regional lymphadenopathy. Death is the frequent outcome of the disease even though it is radiosensitive.
- Transitional cell epidermoid carcinoma are chiefly from the tonsil, base of the tongue and nasopharynx due to presence of that type of epithelium. This is extremely malignant and causes death soon.

Clinical Features

- Primary lesion is very small, often completely hidden, usually slightly elevated and either frankly ulcerated or presenting a granular eroded surface.
- Tumor is indurated and in some instances appears as an exophytic or fungating growth.
- Swelling of the regional lymph node is most common followed by sore throat, nasal obstruction, defective hearing or ear pain, headache, dysphagia, epistaxis and ocular symptoms.

Histologic Features

- Transitional cell epidermoid carcinoma consists of cells growing in solid sheets or in cords and nests.
- Individual cells are moderately large, round or polyhedral and exhibit a lightly basophilic cytoplasm and indistinct cell outline.
- Nuclei appears large and round and exhibit mitotic activity. Keratinization and pear formation are completely lacking.
- Stroma exhibits little or no lymphocytic infiltration.
- Lymphoepithelioma is made up of cells growing in a syncytial pattern with the stroma infiltrated by varying numbers of lymphocytes.
- Individual cells are large and polyhedral with indistinct outlines.
- Cytoplasm stains lightly eosinophilic. Nuclei appear large, oval and vesicular and characteristically contain one or two large eosinophilic nucleoli.

Treatment and Prognosis

- Because of radio sensitivity, X-ray radiation has been the most commonly accepted treatment.
- Complicating factor is widespread of metastatic organ.
- Unfavorable prognosis can be readily understood.

MALIGNANT MELANOMA

- It is a neoplasm of epidermal melanocytes.
- One of the more biologically unpredictable and deadly of all human neoplasms.
- Third most common cancer of the skin.
- Epidemiologic studies have supported that sunlight is an important etiologic factor in cutaneous melanoma.
- It has been seen that two phases of growth is seen
 - Radial growth phase
 - Vertical growth phase.
- Radial growth phase is the initial phase, which may last many years. The neoplastic process is confined to the epidermis.
- Neoplastic cell are shed with normally maturing epithelial cells and although some neoplastic cell may actually penetrate the basement membrane, they are destroyed by a host cell immunologic response.

- The vertical growth phase begins when neoplastic cell populate the underlying dermis. This takes place because of increased virulence of the neoplastic cells, an increased host-cell response or a combination of both.
- Metastasis is possible once the melanoma enters the vertical growth phase. It is recognized that both the phases are not seen in all cases.
- Nodular melanoma exists only in the vertical growth phase.
- Cutaneous melanoma has been classified into a number of types.
- Superficial spreading melanoma.
- Nodular melanoma.
- Lentigo maligna melanoma.

Clinical Features

- Superficial spreading type is most common. It exists in a radial growth phase which has been called “pre-malignant melanosis” or pagetoid melanoma”.
- Lesions present as a tan, brown, black or admixed lesion on sun-exposed skin, especially the back. Also occurs on skin of the head and neck, chest and abdomen and the extremities.
- Radial growth phase may last for several months to years. Vertical growth phase is characterized by in size, change in color, nodularity and at times ulceration.
- Nodular melanoma has no clinically recognizable radial-growth phase, existing solely in a vertical growth phase.
- Presents as a sharply delineated nodule with degrees of pigmentation. May be pink or black. They have predilection for occurrence on back and head and neck skin of men.
- Lentigo maligna melanoma accounts for approximately 10 percent of cutaneous melanomas.
- It exists in a radial-growth phase which is known as “lentigo maligna” or “melanotic freckle of Hutchinson”.
- More common in woman than men. These lesions can remain in the radial- growth phase for years.
- Melanoma may occur as a primary lesion not only on the skin but also in the eye and on mucous membranes.
- Also been reported as a primary lesion in the parotid gland, although melanomas in this site are usually metastatic to lymph nodes in the parotid region.

Oral Manifestation

- Primary oral melanoma is nearly twice as common in men as in women.
- Site → Definite predilection on palate and maxillary gingiva/ alveolar ridge.
- Also buccal mucosa, gingiva, tongue, lip and floor of the mouth.
- Lesions appear as a deeply pigmented area, at time ulcerated and hemorrhagic which increase in size later.

Histologic Features

- Intraepithelial component of superficial spreading melanoma is characterized by the presence of large, epitheloid melanocytes distributed in so-called. "Pagetoid" manner

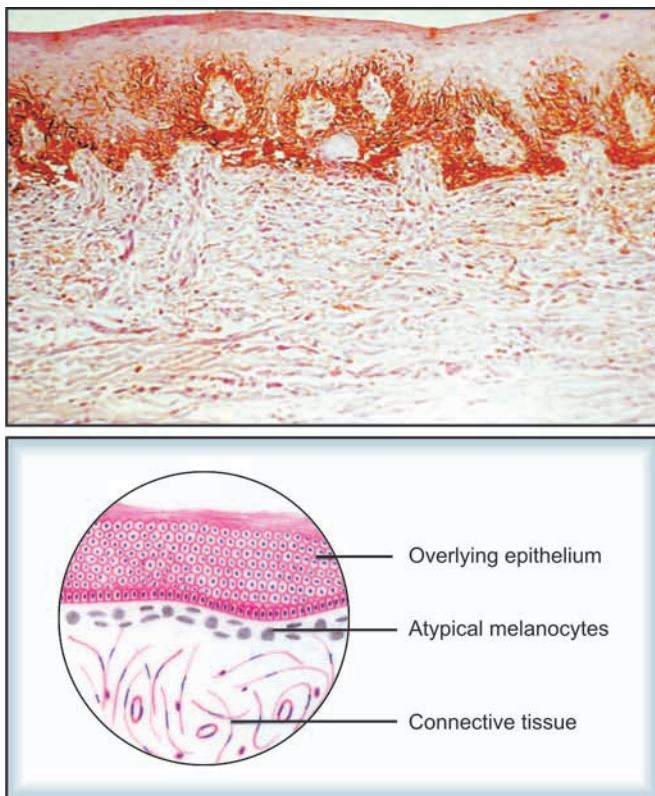


Fig. 12.14: Malignant melanoma

- Macrophages and melanophages may be present.
- Cells may be arranged singly or in clusters melanin pigments is usually scanty.
- Lentigo maligna melanoma is characterized by number of typical melanocytes within the basal epithelial layer.
- Epithelium is atrophic and the dermal collagen shows the effects of sun damage, i.e. basophilic degradation.
- Invasive spindle shaped cells seen into underlying dermis.

Treatment and Prognosis

- Surgical excision.
- Elective lymph node dissection.
- Tumor > 0.75 mm in thickness and located in “BANS” have a greater tendency to metastasize.
- Chemotherapy, immunotherapy and radiation therapy have been used in the treatment of cutaneous melanoma.
- Oral melanoma → surgical excision.
- Women have much better survival rate up to 50 years then decreases.
- Melanomas of skin have more favorable prognosis.
- Nodular have much better prognosis than lentigo maligna melanoma.

BENIGN TUMORS OF THE MESENCHYMAL TISSUE ORIGIN

FIBROMA

- It is a benign neoplasm of the fibrous connective tissue.
- It is characterized by excessive proliferation of the fibroblast cells with large amount of collagen synthesis.

Clinical Features

- Seen in third to fifth decade of life.
- More common among females.
- Most commonly seen in relation to the gingiva and other sites include buccal mucosa, tongue, lip and palate.
- Usually, presents as a small, well-circumscribed, slowly growing, painless nodule, having a pedunculated or sessile base.

- The surface of the lesion is usually smooth and soft or firm in consistency. In most of the cases, the overlying epithelium appears normal but stretched.
- In case of trauma or injury the lesion may become painful and surface ulceration is seen.

Histologic Features

- Excessive proliferation of large number of spindle-shaped fibroblasts or fibrocytes, which can produce bundles of interlacing collagen fibers within the lesion.
- Overlying epithelium is thin and there is flattening of its rete pegs. Connective tissue stroma is relatively avascular and may contain small calcified or ossified masses occasionally.
- Few chronic inflammatory cell infiltration within the stroma is seen.

Treatment

- Surgical excision.

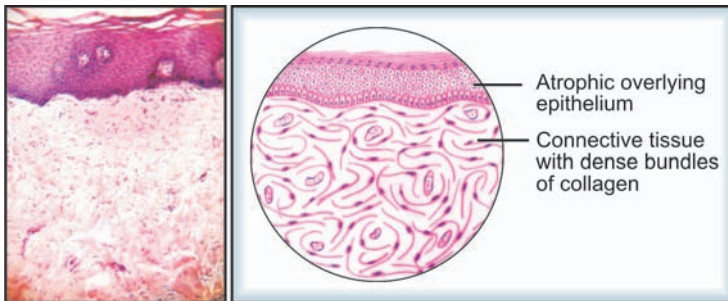


Fig. 12.15: Fibroma

OSSIFYING FIBROMA

- It is a benign neoplasm of the oral cavity that occurs either in relation to the soft tissue or within the jaw bone.
- The soft tissue lesions developing outside the bone are called peripheral ossifying fibromas and lesions those occurring inside the bone are known as central ossifying fibromas.

Clinical Features

- Mostly occurs in young adults.
- More common among females.
- The peripheral type mostly occurs in gingiva, whereas the central lesion frequently involves the mandibular bone.
- It is a slowly enlarging, small, painless swelling, causing mild deformity and displacement of the regional teeth.
- Overlying covering epithelium is normal in appearance.
- If the lesions are very aggressive in nature they produce a very fast enlarging swelling of the bone, with expansion and distorting of the cortical plates. May cause difficulty in speech and taking food.

Radiological Features

- Radiologically a central ossifying fibroma produces a small, circumscribed radiolucent area which is well demarcated from the surrounding normal bone.
- This feature helps in differentiating between the ossifying fibroma and fibrous dysplasia because radiologically the fibrous dysplasia are not well demarcated lesions and are gradually blended with surrounding normal bone.
- Within the radiolucent area multiple calcified, radiopaque areas of varying radiodensity are found in case of ossifying fibroma.
- Displacement of regional teeth is seen.

Histologic Features

- The most important histological difference between the central and peripheral ossifying fibroma is that in case of peripheral variety the covering epithelium will be present and it is absent in the central variety. The rest of the features are almost identical in both cases.
- Multiple numbers of proliferating fibroblasts within a delicate connective tissue stroma, consisting of interlacing collagen fibers and many blood capillaries is also seen.
- Multiple small foci of irregular bone formation are seen within the connective tissue stroma.
- The lesion is surrounded by a capsule.

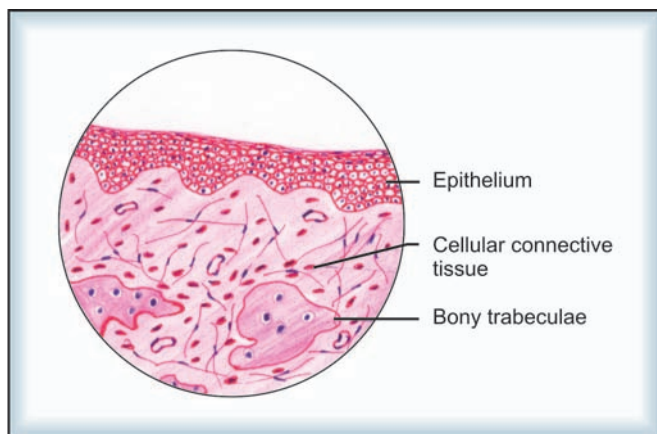


Fig. 12.16: Peripheral ossifying fibroma

Differential Diagnosis

- Fibrous dysplasia.
- Cementifying fibroma.
- Condensing osteitis.

Treatment

- By surgical excision.

PERIPHERAL GIANT CELL GRANULOMA

- These are not true neoplasms but are reactive overgrowths of tissue. Commonly seen in the tooth bearing areas of jaw.

Clinical Features

- Lesion occurs at any age but most commonly in second to third decades.
- More commonly in females.
- Site of involvement is gingiva, alveolar process (tooth bearing areas), between the first molar and the incisor region and on the crest of the edentulous alveolar ridge.
- The mandibular gingival is more commonly affected than the maxillary one. The lesion appears to have originated from the deeper structures like the periodontal ligament or the periosteum of bone.

Presentation

- Peripheral giant cell granuloma appears as a small, well-circumscribed, pedunculated and lobulated, soft tissue growth in the gingival area.
- It often causes displacement of the regional teeth.
- Either dark red or bluish red in color and the covering epithelium is mostly ulcerated.
- Bleeding from the lesion, which may be either spontaneous or upon provocation, is an important features of peripheral giant cell granuloma.

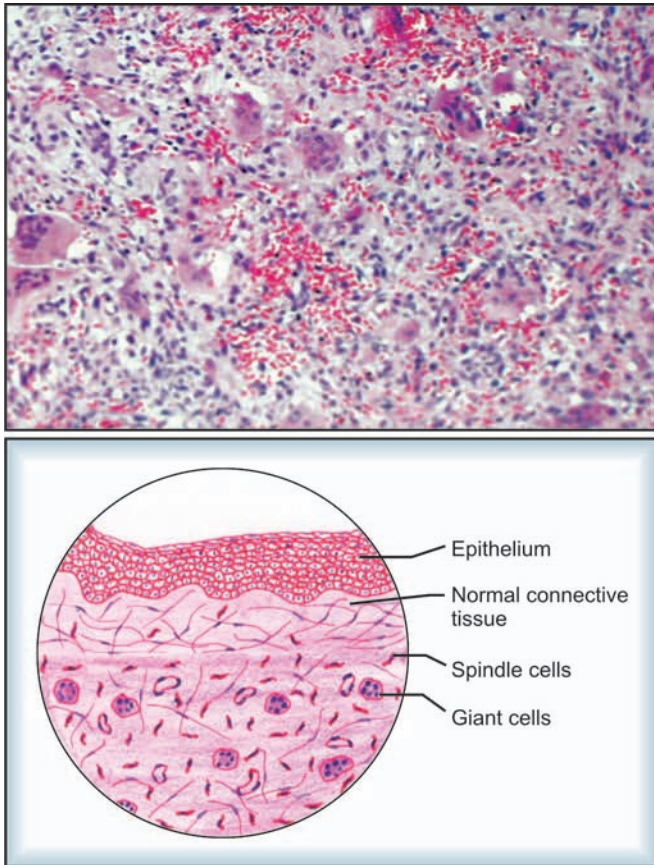


Fig. 12.17: Peripheral giant cell granuloma (giant cell epulis)

- Multiple small areas of blood pigments are also commonly present on the surface of the lesion.

Radiological Features

- Radiologically peripherally it shows superficial erosion of bone, which is commonly known as “peripheral cuffing”.

Histopathology

- Overlying covering epithelium is mostly ulcerated and the underlying connective tissue stroma consists of numerous proliferating fibroblasts, blood capillaries and many multinucleated giant cells.
- Interstitial edema is commonly observed in the connective tissue stroma. Large number of chronic inflammatory cell infiltrations seen.
- The giant cells in the connective tissue stroma always tend to group around the blood capillaries and in few cases; they are present even within the lumen of these capillaries.
- Areas of hemorrhage and hemosiderin pigmentations within the stroma is seen.
- Sometimes, foci of osteoid or bone tissue are present at the periphery of the lesion.

Differential Diagnosis

- Pyogenic granuloma.
- Central giant cell granuloma.
- Fibroepithelial polyp.
- Peripheral ossifying fibroma.
- Fibroma.

Treatment

- By surgical excision including the base, recurrence is common.

CENTRAL GIANT CELL GRANULOMA

- This is a relatively common benign giant cell lesion of the jaw bones. It lesion also occurs in the long bones and in many cases,

is confused with the giant cell tumor of bone. It is to be mentioned clearly that the giant cell granulomas are reactive lesions, while the giant cell tumors are the real neoplastic conditions of the bone.

Etiology

- Exact etiology is not known, few probable causes have been implicated.
- A reparative response to the intrabony hemorrhage or inflammation.
- Some believe this lesion as a truly neoplastic one.
- It may develop as a developmental anomaly and the pathogenesis could be similar to that of the aneurysmal bone cyst.

Clinical Features

- Seen in 10 - 20 years of age.
- Slightly more among females.
- Mostly involves mandible and are little uncommon in the maxilla.
- Tooth bearing areas of the jaw especially in the anterior part, are most frequently affected, and few lesions even cross the midline.
- A slow enlarging, bony hard swelling of the jaw, which is often painful on palpation.
- Expansion of the cortical plates with loosening and exfoliation of the regional teeth is common.
- Anesthesia or paresthesia of the mental nerve occurs sometimes.
- Few lesions cause perforation of the cortical plates, resulting in protrusion of the tumor as a dome-shaped purplish nodule on the alveolar ridge.

Radiological Features

- On the radiograph mostly appears as multilocular radiolucent area, having well-defined scalloped border.
- Sometimes, it can present an unilocular drop-shaped radiolucency.
- Expansion and thinning of the cortical plates or even its perforation is common.
- Roots of the regional teeth are displaced and resorbed in few cases.

Histopathology

- Lesion consists of a highly vascular connective tissue stroma, containing numerous proliferating fibroblasts and variable amount of interlacing collagen fibers.
- Numerous small blood vessels, with areas of hemorrhage and hemosiderin pigmentations are found within the connective tissue.
- Multiple multinucleated giant cells of varying size are present, which are irregularly distributed within the connective tissue stroma.
- In few cases the giant cells are frequently aggregated around the blood vessels.
- Few small foci of osteoid may also be present.
- Minimum amount of chronic inflammatory cell infiltration within the stroma.

Differential Diagnosis

- Ameloblastoma.
- Odontogenic myxoma.
- Odontogenic keratocyst.
- Aneurysmal bone cyst.
- Hyperparathyroidism.
- Giant cell tumor of bone (osteoclastoma).

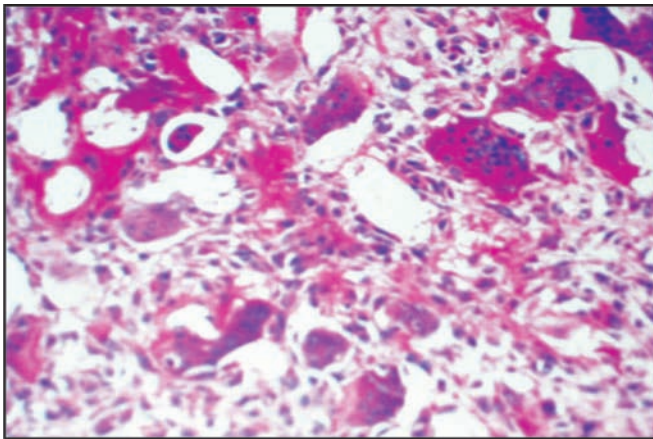


Fig. 12.18: Central giant cell granuloma

Treatment

- Surgical excision and thorough curettage.

LIPOMA

- Its a rare benign neoplasm of the oral cavity which is arising from the mature fat cells.

Clinical Features

- Mostly seen among adults and usually presents a slow growing, small, very soft, lobulated and pedunculated swelling that often gives a cystic feeling.

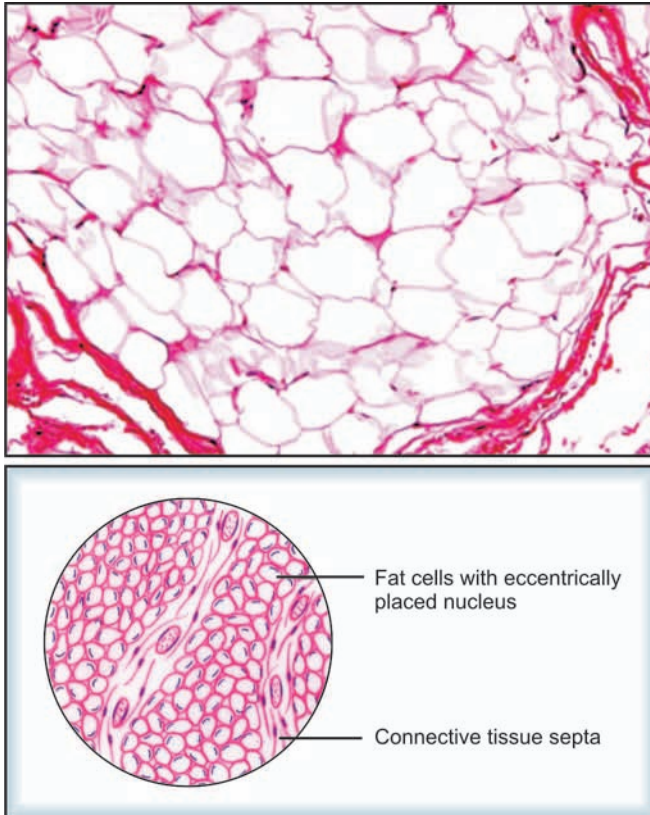


Fig. 12.19: Lipoma

- Usually yellowish in color and is mostly arising from the buccal pad of fat, tongue or salivary gland tissue.
- Microscopically lipoma consists of proliferating mature fat cells within a loose areolar tissue, being surrounded by a fibrous capsule.

Treatment

- By surgical excision.

HEMANGIOMA

- Common benign tumor arising from the vascular tissue and is characterized by the proliferation of numerous blood capillaries.
- Sometimes considered as a congenital lesion.

Clinical Features

- Most of the hemangiomas are present at birth or they arise at an early age.
- It is more commonly seen among females.
- Site of involvement is intraoral lip, tongue, buccal mucosa and palate.
- The tumor also frequently develops inside the jaw bones as a central lesion and in few cases, it may occur intramuscularly too.
- Clinically represents as a flat or raised, nodular or diffuse swelling having a deep red or bluish red color.
- On palpation, it is soft and pulsatile and upon pressure there is a reduction in the size of the tumor as well as disappearance of its color.
- When the pressure is released, the color and the size of the tumor return back, due to the refilling of the tumor with blood.
- If the tumor is connected with a large vessel, “bruits” can be heard from the tumor during auscultation and this is a very important diagnostic clue for hemangioma.

- Trauma or damage to the covering skin or epithelium causes excessive, uncontrolled bleeding from the tumor.
- Intramuscular hemangioma produces a diffuse pulsatile swelling, with pain and limitations of movement of the involved muscle. During examination, the muscular tumor can be moved across the long axis of the muscle but not along it.
- Central hemangioma arising from either the maxilla or the mandible produces a painless, expansile, bony hard swelling with displacement of the regional teeth.

Radiological Features

- On radiograph, central hemangiomas within the maxilla or mandible present multilocular radiolucent areas, with a typical “soap bubble” or “sunburst” appearance. In few cases, the bone destruction may be very extensive with thinning or even perforation of the cortical plates.

Histopathology

- Several histological types of hemangiomas found in the oral cavity and the two main histological types which are commonly seen are the:
 1. Capillary hemangioma, and
 2. Cavernous hemangioma.

CAPILLARY HEMANGIOMA

- It consists of numerous small, proliferating young blood capillaries, which are lined by a single layer of well-formed endothelial cells and are supported by a connective tissue stroma.
- Histologically, capillary hemangioma is confused with other lesions like pyogenic granuloma or a young granulation tissue.
- Interstitial edema and chronic inflammatory cell infiltration is rare in case of hemangioma, but these are otherwise very common in the later two conditions.

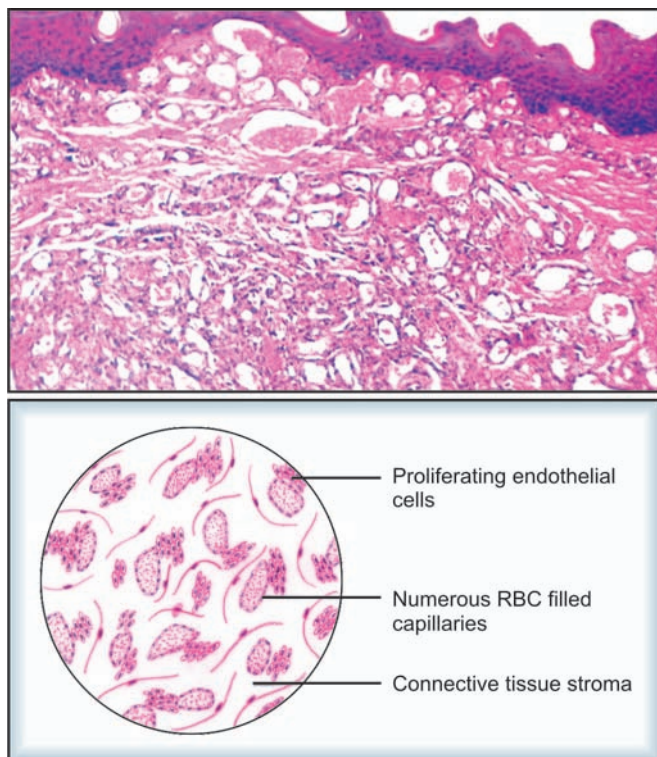


Fig. 12.20: Capillary hemangioma

CAVERNOUS HEMANGIOMAS

- Represents few large, dilated, blood pooled sinuses, which are intercommunicating with one another and these are usually lined by a single layer of flattened endothelial cells.
- Large areas of hemorrhage and hemosiderin pigmentations are often seen within the connective tissue stroma.
- Inflammatory cell infiltration is very rare.
- Laboratory diagnosis by carotid angiogram.

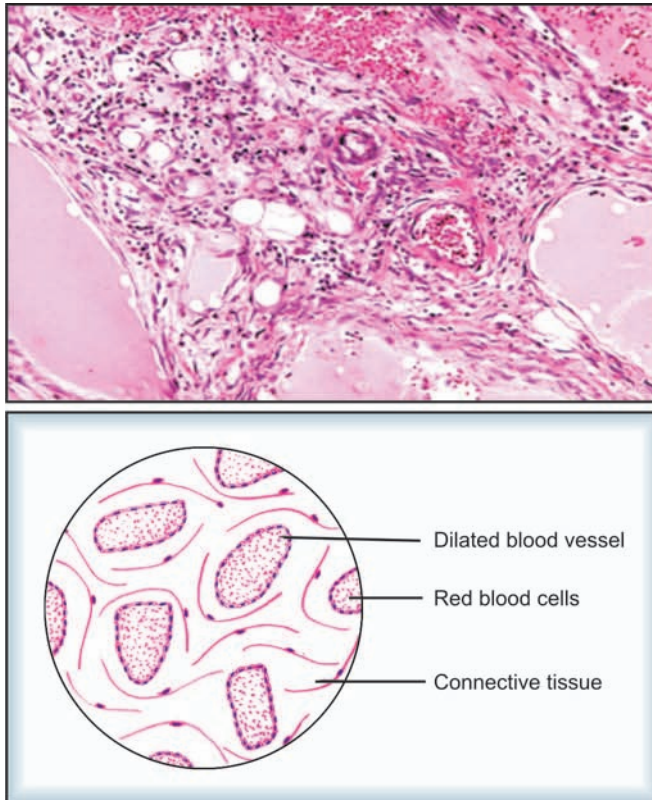


Fig. 12.21: Cavernous hemangioma

Treatment

- Many show spontaneous regression with time.
- Persistent small lesions are treated by conventional or laser surgery or cryosurgery.
- Larger lesions are treated first by the repeated injections of sclerosing agents (e.g. 1% sodium tetradecyl sulfate, or boiling distilled water) inside the lesion for few days. This will cause degeneration of the blood vessels within the tumor with subsequent fibrosis, thereby, making the surgical excision easier and uneventful. However, in any case hemangioma is a dangerous tumor to handle with.

LYMPHANGIOMA

- Lymphangiomas are benign tumors of the lymphatic vessels and are quite uncommon lesions in the oral cavity.

Clinical Features

- Mostly present at birth or develops before the age of 10 years.
- Both sexes are equally affected.
- The tumor most commonly develops in the tongue, but also develops in relation to the palate, buccal mucosa, gingival and lip, etc.
- It is a superficial lymphangioma usually presents a painless, nodular or vesicle-like swelling on the oral mucosa.

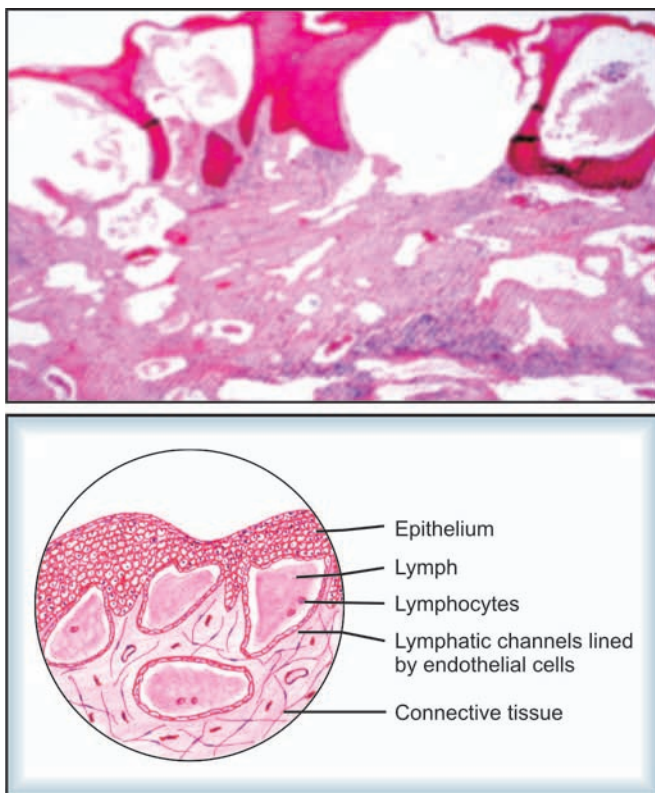


Fig. 12.22: Lymphangioma

- Deeper lesion of lymphangioma produces a diffuse, painless, submucosal lump.
- The color of the lesion is usually lighter than that of the surrounding normal mucosa, but on few occasions, the lymphangioma may show a red-blue discoloration of the surface.
- On palpation, the tumor produces a “crepitates sound” – as the intralesional lymphatic fluids are pushed from one area to the other.
- Lymphangioma of the tongue very often causes macroglossia with indentations of the lateral margin of the tongue by the teeth.
- The lip lesions of lymphangioma often cause diffuse swelling.

Histologic Features

Microscopically lymphangioma reveals the following features:

- There will be diffuse proliferation of multiple numbers of lymphatic vessels within the submucosal tissue. These vessels are usually lined by an endothelial lining and there is presence of homogenous eosinophilic lymph within the lumen of the vessels.
- The tumor is often presented directly adjacent to the overlying epithelium with no intervening connective tissue in between.

Differential Diagnosis

- Hemangioma
- Mucocele
- Large mucosal bullae
- Treatment
- By surgical excision.

MYXOMA

- Myxomas are benign tumors of myxomatous tissue origin, and they can be of either odontogenic or nonodontogenic variety.
- The odontogenic myxomas arise exclusively in the jaw bones, while the nonodontogenic myxomas may occur in any part of the skeleton or soft tissues.
- Clinically myxomas produce slow enlarging, exophytic jaw swelling, with loosening and displacement of teeth. Myxoma

can occur at any age and there is no sex predilection. Both jaws are affected with almost equal frequency.

- Radiologically these lesions produce either unilocular or multilocular radiolucent areas, with a “soap-bubble” appearance. Expansion and distortion of the cortical plates are also commonly observed.
- Histological sections of myxoma tissue reveal the presence of numerous stellate cells within a connective tissue stroma. These cells are known as “myxoblasts” and they produce large amount of glycosaminoglycans, - a mucoid material which is mostly responsible for the gelatinous consistency of the connective tissue stroma in this tumor. Because of the gelatinous consistency of the connective tissue and the infiltrative nature of its cells the tumor becomes very aggressive in nature.
- Treatment of this tumor is usually done by radical surgery, otherwise recurrence is most likely.

CHONDROMA

- Chondromas are benign tumors of cartilaginous tissue origin and are consisting of mature chondrocytes.

Clinical Features

- Elderly people are usually affected, nearly at the age of 50 years.
- Both sexes are almost equally affected. Site nasal septum, anterior part of the maxilla, body and symphysis of the mandible, including the coronoid and the condylar processes.

Presentation

- The tumor presents a slow enlarging, painless, bony hard swelling of the jaw, with expansion and distortion of the cortical plates. In most of the cases, the overlying mucosa appears normal unless it is traumatized.

Radiology

- The tumor radiologically presents an irregular radiolucent area, with foci of calcifications within it. Resorption of the roots of the adjacent teeth are commonly found.

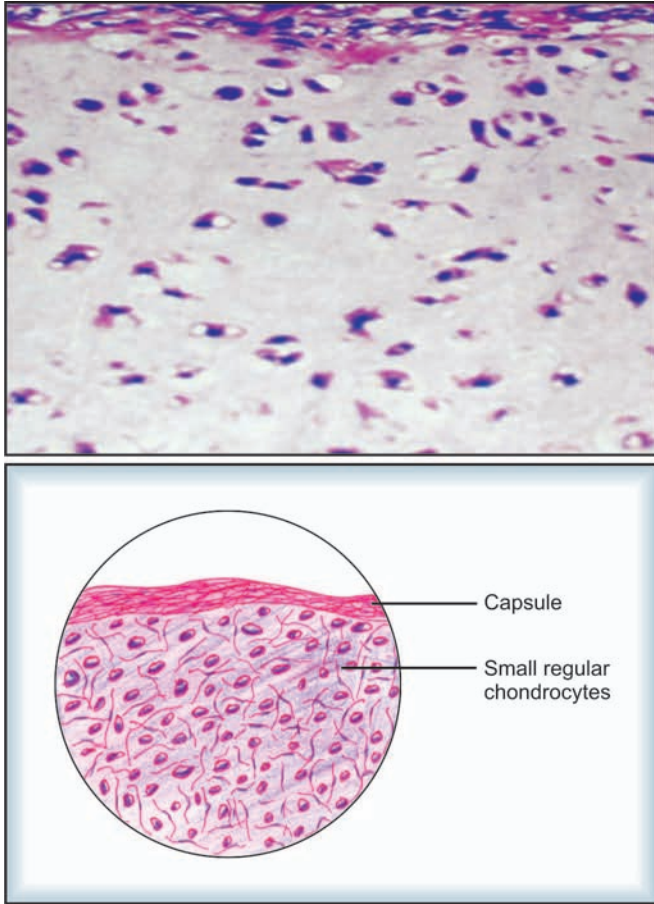


Fig. 12.23: Chondroma

Histopathology

- The lesion consists of well-defined lobules of hyaline cartilage, containing multiple mature chondrocytes. These cells are small in size and they have single, regular nuclei. There are many areas of calcifications as well as areas of necrosis and hemorrhage found within the tumor.

Treatment

- By surgical excisions.

CHONDROBLASTOMA

- Chondroblastoma is a rare benign tumor arising from the epiphyseal regions of the long bones and are usually seen in the mandibular condylar regions infrequently. Most of the patients are aged below 25 years. Clinical and radiological features of chondroblastoma are not usually pathognomonic. Microscopically the tumor shows a highly cellular structure, consisting of numerous round or polyhedral chondroblast like cells in a scanty stroma. The stroma also shows areas of calcifications, along with many multinucleated giant cells. Surgical excision is the usually recommended treatment.

LEIOMYOMA

- Leiomyomas are rare benign tumors arising from the smooth muscle cells of the vascular tissue. Tongue is the most common site for this tumor in the oral cavity and the lesion usually shows a slow growing, painless swelling. In most of the cases, adult people are affected by this tumor.
- Microscopy reveals typical bundles of smooth muscle fibers with interlacing bundles of collagen. The muscle cells are typically spindle-shaped with a prominent nucleus and the cells also show intracytoplasmic myofibrils.
- Van-Gieson staining may be used to differentiate between the collagen fibers (red), from the muscle fibers (yellow), within the tumor. Sometimes, groups of smooth muscle fibers are surrounding the periphery of a blood vessel which indicates the vascular origin of the tumor itself. Treatment is done by surgical excision.

RHABDOMYOMA

Rhabdomyomas are benign tumors arising from the striated muscles and are extremely rare in the oral cavity (most common site of occurrence is the heart muscles). However, intraoral tumors usually occur on the tongue or the floor of the mouth and are more common among males, with a peak age of occurrence being the fifth decade of life. Clinically the tumor presents a slow growing, well-circumscribed, painless mass. Microscopically the tumor presents a sharply

outlined, unencapsulated mass, consisting of larger striated muscle cells showing granular eosinophilic cytoplasm. Multiple vacuoles are often seen within the cell cytoplasm and are mostly confused as fatty spaces. These vacuoles often give the cell cytoplasm a spiderly outline, which also occasionally shows cross-striations. Surgical excision is the treatment of choice.

OSTEOMA

Definition

- Osteomas are benign tumors arising from the osseous (bone) tissue and are consisting of either mature compact or cancellous bone.

Clinical Features

- Age → Second to fifth decade of life.
- Sex → More in females.
- Site → Both maxilla and mandible are affected, and the lesion arises either endosteally or periosteally. On rare occasions, this tumor may arise from the soft tissues like the tongue or buccal mucosa, etc.

Presentation

- The osteomas present slow enlarging, small, asymptomatic, bony hard swelling of the jaw. The tumor sometimes produces expansion of the cortical plates with displacement of the regional teeth. Multiple osteomas are sometimes present in relation to the Gardner's syndrome, which includes intestinal polyposis, multiple osteomas of bone, desmoids fibromas of skin, epidermoid cysts and many impacted permanent or supernumerary teeth.

Radiology

- Both the periosteal and the endosteal forms of osteomas of the jaw bones radiographically produce a well-circumscribed, sclerotic area in the affected bone.

Histopathology

- Osteomas histologically present two distinct forms in one form, the lesion is composed of a relatively dense, compact bone, with minimum marrow spaces, and in other form of the disease, and it consists of multiple trabeculae of cancellous bone, with large amount of fibrofatty marrow spaces.

Differential Diagnosis

- Exostoses
- Osteoblastoma
- Sclerosing osteomyelitis
- Odontomas.

Treatment

- Treatment by surgical excision.
- Osteoblastoma and osteoid osteoma.
- Osteoblastoma and osteoid osteoma are two benign tumors of bone and are generally believed to be the variants of a single tumor. They are found mostly in the long bones and are usually rare in the jaws. Clinically, both these tumors cause pain and swelling with expansion of the cortical plates, although pain is a relatively more common feature for the osteoid osteoma. Usually, the later lesions are less than 1 cm in diameter and their radiographs show a central area of radiolucency, which is surrounded by a peripheral osteosclerotic zone. Histologically the osteoid osteoma presents a vascular stroma, within which multiple numbers of immature osteoid trabeculae are found. These bony trabeculae are always lined by many osteoblasts and few osteoclasts.
- On the other hand, osteoblastomas measure more than 1 cm in diameter in most of the cases and produce a radiolucent area inside the bone, with no peripheral sclerotic margin. Microscopy shows a vascular stroma, containing broad, widely spaced osteoids and primitive bone, which are surrounded by many osteoblasts and few osteoclasts. Some areas of hemorrhage are also present. The histological differentiation of the osteoid

osteoma and the osteoblastoma from the osteosarcoma may sometimes be difficult. The treatment is done by surgical excision in both the cases.

GRANULAR CELL MYOBLASTOMA

- Recent name is “granular cell tumor”. This lesion was earlier considered as a degenerative disease of muscle, but nowadays on the basis of the ultrastructural studies and especially due to the presence of S-100 protein, it is being considered as a tumor of Schwann cell origin.

Clinical Features

- Granular cell myoblastomas are generally uncommon tumors. Tongue is the most frequent site for its occurrence and adult people aged between 30-60 years are mainly affected.

Presentation

- The tumor appears as a small, well-circumscribed lump or mass just beneath the normal epithelium. Sometimes they are multiple in numbers or may be very large in size and are thereby often confused clinically as a carcinoma. The midline lesions of granular cell myoblastoma of the tongue are also frequently mistaken for median rhomboid glossitis.

Histopathology

- This unencapsulated tumor consists of multiple sheets of large polygonal cells, containing pale granular cytoplasm and small compact nuclei.
- The tumor cells are often seen to be lying in continuity with the muscle fibers and thereby severely contradicting their neural origin.
- The overlying epithelium often shows pseudoepitheliomatous hyperplasia which is a very important feature of this tumor.
- Chronic inflammatory cell infiltration is absent in the stroma.

Differential Diagnosis

- Neurofibroma
- Schwannoma

- Traumatic fibroma
- Salivary gland tumors
- Squamous cell carcinoma.

Treatment

- By surgical excision.

SCHWANNOMA

(Neurilemmoma)

Definition

- Schwannoma or neurilemmoma is the benign neoplasm derived from the Schwann cells (neural crest origin) of the neurilemmoma (a structure that surrounds the peripheral nerves).

Clinical Features

- Age → The tumor can arise at any age.
- Sex → Males and females are equally affected.
- Site → Intraorally, tongue is the most favored location for this tumor which is followed by palate, floor of the mouth, buccal mucosa, lip and jaw bones (chiefly mandible).

Presentation

- Schwannoma usually presents a slow enlarging, well-circumscribed, painless nodule of varying size.
- In some cases, the tumor enlarges at a very faster rate, with accompanying pain and paresthesia.
- Bony tumors appear as serious lesions, causing considerable expansion and destruction of the bone, with mobility and displacement of the regional teeth.

Radiology

- Radiographically intraosseous schwannomas mostly produce multilocular radiolucency, with severe expansion and destruction of the cortical plates.

Histopathology

- Histopathological features of the tumor are highly characteristic.
- The tumor is encapsulated and it shows proliferating spindle-shaped cells (neoplastic Schwann cell), arranged in two different patterns: Antoni-A and Antoni-B.
- The Antoni-A areas consist of spindle-shaped cells, arranged in palisading waves and the cells are surrounded by an acellular eosinophilic zone called “Verocay body”.
- Antoni-B areas show the spindle-shaped cells arranged in a haphazard manner, and there is no palisading arrangement.
- Tumor cells of schwannoma have very little tendency for malignant transformation.
- The main nerve, with which the tumor is associated, is often pushed aside and does not become enmassed within the tumor itself.

Treatment

- By surgical excision.

NEUROFIBROMA

- Neurofibroma is a benign neural tissue tumor, arising from the perineural fibroblasts.
- Neurofibroma may occur either as a single tumor or there may be multiple lesions occurring at a time as a part of the syndrome called “neurofibromatosis”.

Clinical Features

- The solitary neurofibroma develops at any age and presents a small, asymptomatic, submucosal mass, with multiple small lobulation on the surface. The tongue, buccal mucosa and vestibule are the common sites for this tumor.
- Solitary neurofibroma is mostly an innocuous lesion but when it occurs as a part of the syndrome-neurofibromatosis, it shows a higher tendency for malignant transformation (neurogenic sarcoma).
- Neurofibromatosis on the other hand is a common syndrome and it consists of the features like – multiple solitary neuro-

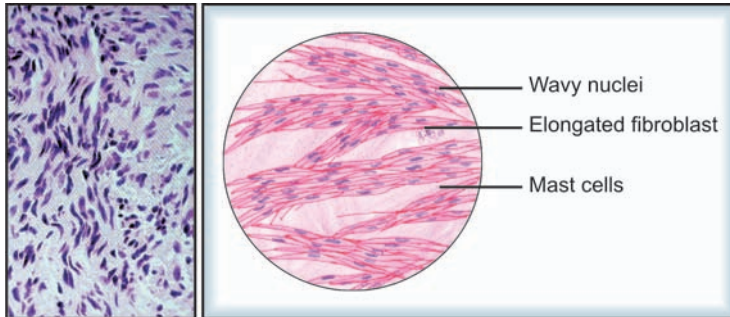


Fig. 12.24: Neurofibroma

fibromas, café au lait spots, bone abnormality and central nervous system defects, etc.

Histopathology

- Histologically neurofibroma, be a solitary lesion or multiple lesions as seen in case of neurofibromatosis, always presents the similar appearance.
- The features include numerous proliferating spindle-shaped tumor cells (neurofibroblast); these cells have fusiform or wavy nuclei.
- The connective tissue stroma may have a myxoid appearance and it shows many mast cells in addition to the neurofibroblasts.

Treatment

- By surgical excision.

MALIGNANT TUMORS OF MESENCHYMAL TISSUE ORIGIN

FIBROSARCOMA

- Fibrosarcomas are relatively uncommon malignant tumors arising from the fibrous connective tissue and are characterized by an increased abnormal proliferation of malignant fibroblast cells.

Clinical Features

- Age → The tumor can occur at any age, but the peak age of occurrence is 35 to 55 years.
- Sex → Both sexes are almost equally affected.
- Site → The common intraoral sites for fibrosarcoma are cheek, maxillary sinus, palate, pharynx and maxilla or mandible. When fibrosarcoma occurs in relation to the bone, it arises either periosteally or endosteally.
- In the initial phase, the tumor is mostly asymptomatic and it resembles a benign fibrous overgrowth.
- In the advanced stage, the tumor starts growing at a very faster rate and gives rise to a large, bulky, firm lobulated “fleshy” mass.

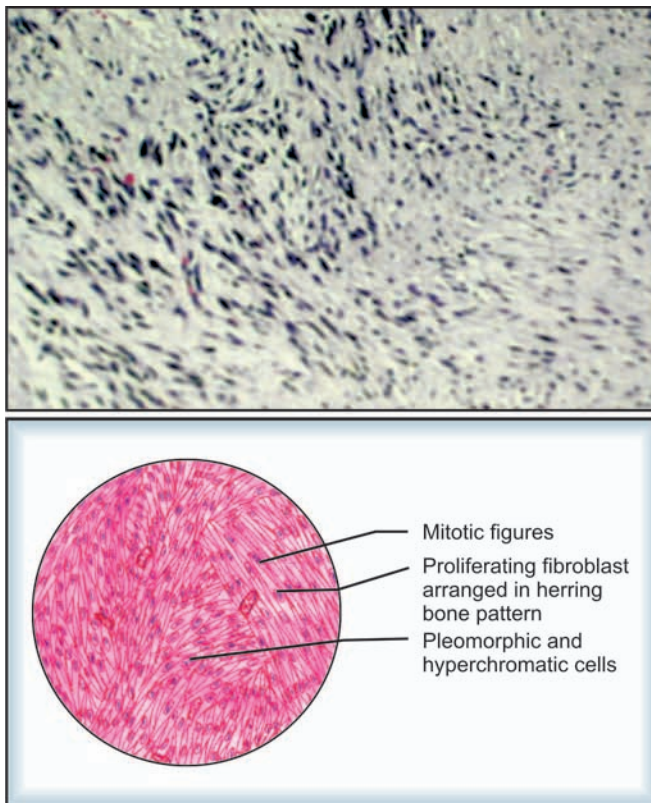


Fig. 12.25: Fibrosarcoma

- Although the lesions usually have a smooth surface but ulceration, pain and secondary infections, etc. often develop.
- The intrabony lesions of fibrosarcoma produce severe destruction of the bone, with loosening and exfoliation of regional teeth.
- Most of the patients appear to be severally ill, with marked deterioration of the general health.

Histologic Features

- Histologically the tumor presents actively proliferating, numerous spindle-shaped malignant fibroblast cells in a connective tissue stroma and these cells often have “tadpole”-like appearance.
- Most of these malignant cells are very well differentiated and each cell contains a large, uniformly stained, elongated, hyperchromatic nucleus with a scanty cytoplasm.
- Within the stroma, these malignant fibroblast cells typically spread in a “streaming fashion” and there is very minimum amount of collagen synthesis by them.
- In many cases, the tumor cells show abnormal mitotic activity in the form of “biradiate” or triradiate mitosis.

Treatment

- Radical surgical excision and chemotherapy. Radiotherapy is not effective. Prognosis is surprisingly good, as the tumor rarely shows distant metastasis.

MALIGNANT FIBROUS HISTIOCYTOMA

- Malignant fibrous histiocytomas are a group of malignant tumors with a very aggressive course.

Clinical Features

- These tumors are not uncommon in the oral cavity and they predominantly affect the young people. The maxillary or mandibular bones, tongue, buccal mucosa and nasopharynx, etc: are the common sites of occurrence. Clinically, these tumors most frequently produce a fast enlarging, lobulated, exophytic, ulcerated lesion or in many cases, they may appear as “fleshy growths” which resemble fibrosarcoma.

- Pain, hemorrhage, anesthesia or paresthesia, etc. may be the accompanying features in most of the lesions.

Histopathology

- Histologically the tumor consists of either polyhedral or oval shaped malignant histiocytes, or spindle-shaped malignant fibroblast-like cells. These cells are generally arranged in a typical “storiform” pattern within the connective tissue stroma.
- Multiple multinucleated giant cells are often present within the tumor and in many cases; the tumor is named as the “giant cell variant” of fibrous histiocytoma if the number of giant cells are extremely high.

Treatment

- Treatment by surgical excision, radiotherapy and chemotherapy.

LIPOSARCOMA

- Liposarcomas are extremely rare malignant tumors of the adipose tissue and are mostly seen among male people of any age. Clinically, the tumor produces a slow growing, firm, lobulated, submucosal mass which is often confused as a large cyst.
- Microscopically liposarcomas are more cellular than the lipomas and they consist of multiple number of proliferating malignant lipoblasts and few “signet-ring cells” (vacuolated lipoblasts). These tumor cells often produce a large amount of fat within the tissue, which often gives the tumor a myxoid appearance. The treatment is done by surgical excision and radiotherapy.
- Hemangiopericytoma and hemangioendothelioma.
- Hemangiopericytoma and hemangioendothelioma are malignant tumors arising from the components of the blood vessels. The cross-section of a normal blood vessel reveals a central ring of reticulin sheath, which is lined on the outer aspect by the spindle-shaped cells called pericytes, while on its inner aspect it is lined by a single layer of endothelial cells.
- When a tumor develops from the pericytes, it is known as hemangiopericytoma and when a tumor develops from the endothelial cells, it is known as hemangioendothelioma. Both

these tumors are generally malignant in nature and they produce large, nodular painful swelling with ulceration on the surface and paresthesia.

- Microscopically, hemangiopericytoma reveals many capillary-like tubules, lined by a single layer of normal appearing endothelial cell in its inner surface, but on the outer surface it is surrounded by many densely packed proliferating spindle-shaped malignant pericytes, which often reveal pronounced cellular pleomorphism and nuclear hyperchromatism. An increased abnormal mitosis is often seen.
- The hemangioendotheliomas, on the other hand, show proliferating malignant endothelial cells within the capillary lumen, which are filling up the entire lumen space and expanding it. In this case, the pericytes situated on the outer aspect of the blood vessel appear absolutely normal. The microscopic differentiation between these two tumors can be made by the use of special stain named reticulin stain, the reticulin-stained sections reveal the presence of pericytes on the outer aspect of reticulin sheath of the blood vessels, whereas the endothelial cells will be found in the hemangiopericytoma and hemangioendothelioma are done by surgery, chemotherapy or radiotherapy.

KAPOSI'S SARCOMA

- Kaposi's sarcoma (KS) is a malignant neoplasm arising from the endothelial cells of the blood vessel.

Etiology

- Several etiologic factors have been suspected for the genesis of this tumor which is as follows.

Genetic Predisposition

- Infection by human immunodeficiency virus (HIV) and cytomegalovirus (CMV).

- Environmental factors.
- Immunosuppression.

Clinical Features

- The Kaposi's sarcoma has three different clinical patterns.
- Classic form of Kaposi's sarcoma: This type of tumor appears as a red-blue, inflamed looking lesion, on the distal lower extremities among the old Mediterranean Jews. Oral lesions in this type are rare.
- Endemic Kaposi's sarcoma: This is mostly seen among the children and young black Africans, and the lesions appearing commonly on the skin, lymph nodes and rarely in the viscera.
- Epidemic Kaposi's sarcoma: These are mostly related to the AIDS patients and they appear as nodular skin or mucosal lesions, which rapidly spread to the lymph nodes and the viscera. Oral lesions are mostly seen in relation to this type and any oral region can be involved but palate is the most common site.
- AIDS patients with the history of homosexuality, develop Kaposi's sarcoma very frequently, although it is not yet clear whether the AIDS related Kaposi's sarcomas occur directly due to the HIV infection or secondary to the immunosuppression.
- The Kaposi's sarcoma usually presents three different clinical stages:
 1. Patch stage
 2. Plaque stage
 3. Nodular stage, all these stages can occur in any form of the disease mentioned earlier.
- *Patch stage*: It is the initial stage and it appears as a pink, red or purple macules on the extremities. Microscopically the "patch" consists of dilated, irregular blood vessels, which are lined by normal appearing endothelial cells and there may be some chronic inflammatory cell infiltration in the connective tissue stroma. This stage histologically resembles pyogenic granuloma very closely.
- *Plaque stage*: With time, the patch stage is converted into the plaque stage and it appears as a large, raised, violaceous plaque that histologically shows dilated, jagged, vascular channels, lined by spindle cells, along with perivascular aggregates of similar

spindle cells. In between the vascular structures, RBC, macrophages, plasma cells, lymphocytes and hemosiderin pigments, etc. are often present.

- *Nodular stage*: It is the last stage of the disease and is characterized by the occurrence of multiple nodular lesions on the skin and mucosa. Microscopically the nodular lesion consists of sheets of spindle-shaped cells, in a background of scattered blood vessels and slit-like spaces containing RBC. Marked hemorrhage, hemosiderin pigmentation, lymphocytes and macrophage infiltration, etc. are also seen. This stage often resembles angiosarcomas during microscopy. The treatment of Kaposi's sarcoma is done by chemotherapy and radiotherapy. Surgery is a difficult proposition.

EWING'S SARCOMA

- This uncommon, round cell sarcoma is a primary destructive bone tumor, which frequently involves the jaw bones. These are probably derived from the endothelial cells of the blood vessels or from the undifferentiated reticuloendothelial cells.

Clinical Features

- Mostly the young people between the age of 5 and 25 years are affected and 60 percent of them are males.
- Mandibular ramus is the most common site for this tumor, with occasional involvement of maxilla. The tumor often metastasizes to the other bones, and jaw lesions are mostly metastatic in origin.
- Pain and swelling of the jaws, facial deformity, destruction of the alveolar bone, loosening of the teeth and surface ulceration are the common clinical findings.

Radiology

- "Moth-eaten" destructive radiolucency of the medulla, cortical erosion and expansion, along with periosteal "onion skin" reaction, etc. are the common radiological findings of this tumor.

Histopathology

- Under microscope, the tumor shows proliferating, closely packed, round cells, with monotonous oval or round nuclei. The tumor cells are forming multiple sheets which are separated from one another by thin fibrous bands containing small blood vessels and chronic inflammatory cells. Increased mitosis with areas of tissue necrosis is also commonly observed.

Differential Diagnosis

- Lymphoma
- Leukemia
- Myeloma
- Chondrosarcoma (mesenchymal)
- Small cell osteosarcoma

Treatment

- By radical surgery and radiotherapy

CHONDROSARCOMA

- Chondrosarcomas are uncommon malignant tumors of the cartilaginous tissue. It involves the mandible very frequently, especially in the premolar-molar region, the symphysis and the condyle or the coronoid process. The maxillary tumors often involve the anterior part of the jaw. Most of the patients are in their fifth and sixth decade of life. Clinically chondrosarcoma presents a painless swelling with expansion of the jaw and loosening of the teeth or poor fitting of the artificial dentures.
- The radiograph shows solitary or multiple, “moth-eaten” radiolucent areas or diffuse radiopaque mass, within the jaw. There may be considerable destruction and expansion of the cortical bone. Microscopically the tumor presents a highly cellular lesion with hyperchromatic, pleomorphic malignant chondrocytes, many of which may be multinucleated. Surgery, radiotherapy and chemotherapy are the usual treatment modalities.

MESENCHYMAL CHONDROSARCOMA

- This variant of chondrosarcoma occurs more commonly in the jaw bones and about one-third of the cases involve extrasketal

soft tissues. The tumor affects the younger people more commonly (second and third decade of life) and there is no sex predilection. Microscopy shows masses of small, round or oval cells, with variable amount of cartilage formation. Increased abnormal mitosis and cellular pleomorphism, etc. are rare in the histologic picture of this tumor. Prognosis is usually poor.

OSTEOSARCOMA

- Osteosarcoma is a common malignant neoplasm arising from the bone and after plasma cell myeloma it is the most common primary bone tumor.

Etiology

- The exact etiology is not known, many of the patients give a history of previous trauma to the particular area, where from the tumor has developed. In many cases radiotherapy may also cause the development of this tumor. Osteosarcomas may also develop from the following pre-existing bone diseases:
 - Paget's disease of bone
 - Fibrous dysplasia
 - Giant cell tumor of bone
 - Osteochondroma
 - Bone infarct
 - Chronic osteomyelitis
 - Osteogenesis imperfecta, etc.

Types of Osteosarcoma

The Osteosarcomas may be of various types and these are listed below:

- According to the location
 - Medullary osteosarcoma
 - Periosteal osteosarcoma
 - Periosteal osteosarcoma (arising from the external surface of bone)
 - Soft tissue osteosarcoma (extraskkeletal).
- According to the radiological findings.
 - Osteolytic type of osteosarcoma

- Osteoblastic type of osteosarcoma
 - Mixed type.
- According to the histology.
 - Osteoblastic type of osteosarcoma
 - Chondroblastic type of osteosarcoma
 - Fibroblastic type of osteosarcoma
 - Telangiectatic type of osteosarcoma
 - Clinical features of osteosarcoma.
- Osteosarcomas account for about 20 percent of all sarcomatous lesions and 5% of them occur in the jaw bones.
- Age → It usually occurs in the younger individuals and the jaw lesion occurs approximately at the mean age of 34 years (1 to 2 decades later than the other skeletal osteosarcomas).
- Sex → Males are affected more frequently than females.
- Site → The tumor most commonly involves the long bones, e.g. lower end of the femur, upper end of the tibia, humerus and fibula, etc. In the jaw bones, maxilla is slightly more commonly affected than the mandible. The maxillary lesions most frequently involve the alveolar ridge area, antrum and in few cases, the palate. The mandibular lesions on the other hand commonly involve the symphysis, angle and ramus area as well as the temporomandibular joint. Extraskkeletal osteosarcomas occur rarely in the oral cavity and they may involve the tongue and the lip.

Clinical Presentation

Clinically osteosarcomas may present the following features:

- A very fast enlarging, painful swelling of the jaw, causing expansion and distortion of the cortical plates with restriction of jaw movement.
- Displacement and loosening of the regional teeth are often seen and sometimes the pain arising from the tumor may be confused with a toothache.
- The mandibular tumors frequently cause numbness of the lower lip and chin regions due to the involvement of the inferior alveolar and the mental nerve.
- The maxillary lesions cause paresthesia of the infraorbital nerve, epistaxis, nasal obstruction, loosening of the teeth and pressure in the eye, etc.

- The overlying skin or mucosa often appears red and inflamed, and careful examination may reveal a vascular prominence in the area.
- Ulceration, hemorrhage, pathological fracture, etc. are the common associated features.

Radiological Features

- The radiological features of osteosarcoma are highly variable.
- In osteolytic type of osteosarcoma, the lesion commonly presents a large, irregular, radiolucent area with a typical “moth-eaten” appearance. Expansion and destruction of the cortical plates are also commonly seen and in most of the cases, the lesion does not produce any new bone.
- In osteoblastic type, multiple, irregular, areas of radiopacities are found within the large radiolucent zone and in many cases, they produce a typical “sun-ray” or a “sunburst” appearance at the periphery of the lesion. This typical appearance may be due to the deposition of newly formed bony trabeculae in a radiating fashion at the periphery of the lesion.
- Early osteosarcomas are characterized by localized symmetric widening of the periodontal ligament space around the regional teeth. This phenomenon occurs as a result of invasion of the tumor cells into the periodontal ligament space and subsequent destruction of the supporting alveolar bone.
- Loss of supporting bone often causes displacement of the regional teeth from their normal position in the jaw.
- In most of the cases, the osteosarcomas present a mixed picture of radiolucency and radiopacity, and many lesions radiologically present the evidence of pathological fractures.

Histopathology

- Histologically osteosarcomas are highly characteristic in nature.
- There will be presence of numerous actively proliferating, spindle-shaped, oval or angular, malignant osteoblast cells, showing cellular pleomorphism and nuclear hyperchromatism.
- Multiple areas of newly formed bone or osteoid tissues are also present within the stroma and these are bordered by the malignant tumor cells at their periphery.

- Increased mitotic activity may be seen in few lesions, making the tumor an extremely cellular one, with a very minimum or no tumor bone formation. Histologically this form of osteosarcoma often resembles a fibrosarcoma.
- In the chondroblastic variant of osteosarcoma, the malignant tumor cells produce a large amount of cartilaginous tissue within the tumor, with scanty amount of bone tissue formation; this variant is commonly seen in the oral cavity and has a better prognosis.
- Osteosarcomas in few cases may be extremely vascular in nature and show multiple numbers of large, poorly formed, blood vessels within the stroma. This type of lesions is often called as telangiectatic type of osteosarcomas.

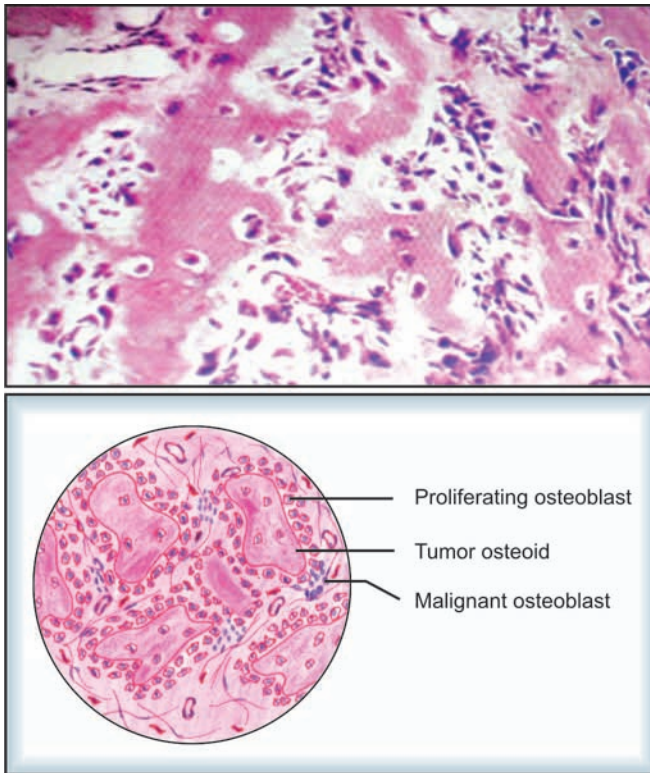


Fig. 12.26: Osteosarcoma

Differential Diagnosis

- Chondrosarcoma
- Fibrosarcoma
- Fracture callus
- Organized hematoma
- Garre's osteomyelitis
- Osteoblastoma
- Eosinophilic granuloma.
(In case of osteosarcoma, both the tissue and the serum alkaline phosphatase level may be raised considerably).

Treatment

- The combination of surgery, radiotherapy and chemotherapy is usually recommended in the treatment of osteosarcoma. Prognosis is usually poor due to the early metastasis of the tumor cells to the lung, brain and other areas. The average five year survival rate is only 10 to 20 percent.

PERIOSTEAL OSTEOSARCOMA

- This uncommon variant of osteosarcoma develops from the external surface of bone and it microscopically presents may well-formed bony trabeculae in a fibrocellular stroma. This tumor does not show the same degree of cellular pleomorphism as seen in case of the endosteal variety. It grows slowly and metastasizes late and thereby has a much better prognosis.

LYMPHOMAS

Definition

- Lymphomas are malignant neoplasms of the cells, native to the lymphoid tissue (i.e. lymphocytes, histiocytes and their precursors and derivatives). Two broad groups of lymphomas have been recognized:
 1. Hodgkin's lymphoma, and
 2. Non-Hodgkin's lymphoma.Although both arise from the lymphoid tissue, the Hodgkin's lymphoma differs from the non-Hodgkin's lymphoma by the presence of Reed-Sternberg giant cells and an increased number

of non-neoplastic inflammatory cells, which frequently outnumber the neoplastic cells (Reed-Sternberg giant cells) in the former lesion.

NON-HODGKIN'S LYMPHOMA

(NHL)

- Non-Hodgkin's lymphoma (NHL) occurs more frequently in the oral cavity than the Hodgkin's lymphoma and the oral NHL lesion shows frequent involvement of the extranodal tissues. Recent literatures also suggest a high incidence of these tumors among AIDS patients.

Clinical Features

- Age → Middle-aged or elderly persons are commonly affected.
- Sex → Slightly more common among males.
- Site → The most common site is the lymphoid tissues of Waldeyer's ring. The other common intraoral sites are the palate, buccal mucosa, tongue, floor of the mouth, retromolar areas and maxilla or mandible.

Clinical Presentation

- Clinically NHL presents the usual features of a sarcomatous lesion.
- The patients may develop some constitutional symptoms like fever, night sweats and weight loss, etc. Along with generalized lymphadenopathy and abdominal pain.
- In the oral cavity, the soft tissue lesions are characterized by a very fast enlarging, exophytic, painful, soft tissue swelling. The overlying surface epithelium appears red and inflamed, and extensive tissue necrosis often causes ulceration, bleeding and superadded candidal infections, etc.
- Intrabony lesions of the jaw produce large, expansile swelling with pain, parasthesia and mobility of the regional teeth. Pathological fracture of the involved bone is also commonly found.
- The jaw lesions of the NHL occur either as central jaw lesions (called the primary lymphoma of bone) or they involve the jaw bones secondarily as an extension from the nearby soft tissue lesions.

Radiological Features

The radiological feature of the NHL of bone reveals the presence of a diffuse, large irregular area of radiolucency with expansion and destruction of the cortical bone. The regional teeth appear to be floating inside the radiolucent zone.

Histopathology

- Histologically NHL is characterized by a monotonous proliferation of malignant lymphocytes, showing cellular pleomorphism and nuclear hyperchromatism, with minimum amount of intervening connective tissue stroma.
- The malignant cells in NHL may be either small in size or uniform in shape, and they can be readily recognized as lymphocytes. In other instances, the tumor cells may be very large and immature in appearance and they often resemble histiocytes.
- These malignant cells, whether large or small, are mostly arranged in two distinct patterns:
 1. Nodular pattern
 2. Diffuse pattern.
- In the nodular pattern, the tumor cells (large or small) tend to aggregate in large clusters, which are separated from one another by very thin connective tissue septa.
- The diffuse pattern of NHL is characterized by monotonous proliferation of tumor cells (large or small) within the connective tissue, with no evidence of cluster formation.
- Special staining with the help of reticulin stain helps in the differentiation between nodular and diffuse type of NHLs.
- In the oral cavity, the diffuse large cell type is the most common form of NHL.
- The simple light microscopy is not always successful in establishing the diagnosis of lymphomas and special investigations like immunohistochemistry, DNA analysis, etc. are always essential.

Treatment

Chemotherapy is the most successful treatment modality in lymphomas. However, radical surgery and radiotherapy are also commonly done.

BURKITT'S LYMPHOMA

- Burkitt's lymphoma is a form of NHL, which occurs commonly, in the jaw bones among the African children. This tumor is believed to be caused by the Epstein-Barr virus and nowadays, this lesion is being reported from other parts of the world as well.

Clinical Features

- Age → Burkitt's lymphoma occurs commonly at the age of about 1 to 3 years.
- Sex → More commonly seen among male children
- Site → The tumor predominantly involves the extranodal areas. Maxilla and mandible are the most frequently affected sites in the head and neck region. The lesion can also develop from the other visceral organs like ovary, kidney, liver and endocrine glands, etc.
- It has been observed that Burkitt's lymphomas occur commonly in the geographic areas, where the malarial infection is very common. The mostly accepted explanation for this phenomenon is that during acute malarial infections, the body's control over the proliferation of "Epstein-Barr virus specific B-lymphocytes" is lost, which results in an increased abnormal neoplastic proliferation of the B-lymphocytes and thereby, leading to the development of Burkitt's lymphoma.

Clinical Presentation

- The tumor begins as a rapidly growing, large, expansile swelling of the jaw with considerable amount of bone destruction and loosening of the teeth.
- Toothache itself is a very frequent complaint among these patients, because of the involvement of the dental pulp by the tumor cells.
- The tumor very frequently involves the mental and the infraorbital nerves and often causing paresthesia of the related structures.
- Involvement of the maxillary sinus (in upper jaw tumors) often results in mobility of the posterior teeth, epistaxis and pressure on the eyeball, etc.

Radiological Features

The radiograph shows presence of a large, irregular, radiolucent area with ill-defined borders which is often giving rise to a “moth-eaten” appearance.

Histopathology

- Histologically Burkitt’s lymphoma is characterized by a monotonous proliferation of undifferentiated, monomorphic, lymphoreticular cells.
- The mitotic activity is abundant and this tumor probably shows the fastest rate of cell multiplication among all malignant lesions.
- Numerous macrophages with an abundant clear cytoplasm, which are often containing cellular debris, are usually found scattered uniformly throughout the tumor and this often produces a very characteristic “starry-sky” appearance.
- Few multinucleated giant cells may also be seen within the tumors,

Treatment

Chemotherapy gives the best response in Burkitt’s lymphoma; of course surgery and radiotherapy are the other therapeutic options.

HODGKIN'S LYMPHOMA

- Hodgkin’s lymphoma (HL) is an extremely rare entity in the oral cavity and in most of the cases; oral structures are involved secondarily, due to the extension of the cervical lymph node tumors.

Clinical Features

- Age → The disease commonly occurs between the age of 15 and 35 years, although it can occur at a higher frequency in the later part of life (beyond 55 years).
- Sex → Slight male predilection is seen.
- Site → The tumor occurs more commonly from the lymphoid tissues of the cervical lymph nodes and the tonsils. Extranodal areas in the oral cavity like the submucosa, maxilla and mandible, etc. are also affected in few cases.

Clinical Presentation

- Persistent generalized lymphadenopathy is the most important feature of HL and the enlarged lymph nodes are firm, rubbery in consistency.
- Generalized weakness, pain in the abdomen and back, weight loss, persistent cough, dyspnea, anorexia, itching of the skin, hemoptysis or melena, and edema, etc. are the other constitutional symptoms.
- Oral lesions of HL show large submucosal swelling with ulceration, pain, paresthesia and fixation to the underlying bone.

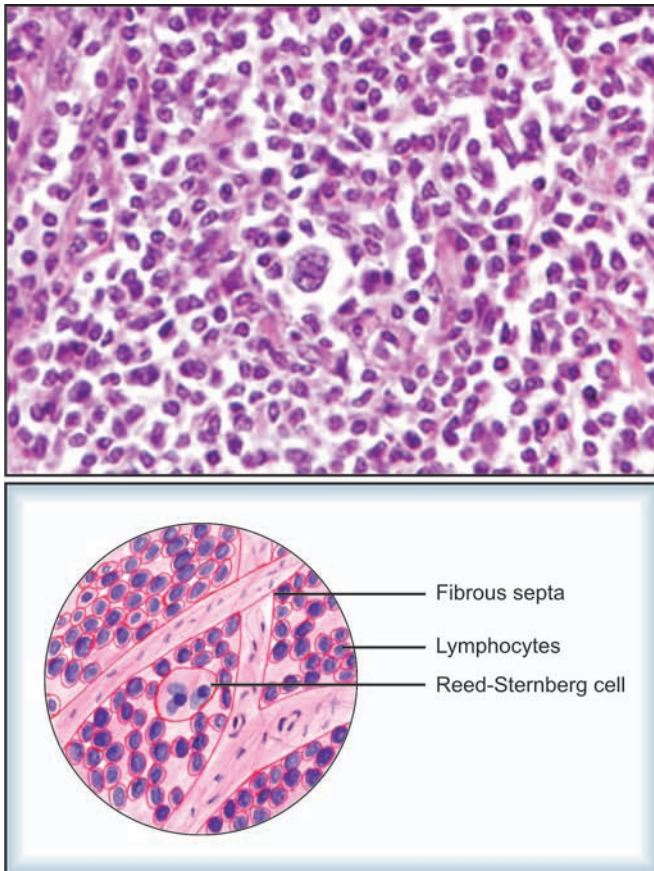


Fig. 12.27: Hodgkin's lymphoma

Histopathology

- In HL, the involved lymph node is histologically characterized by the presence of malignant lymphoid cells and non-neoplastic inflammatory cells, including lymphocytes, macrophages, plasma cells and eosinophils, etc.
- The chief malignant cells of this tumor are the “Reed-Sternberg giant cells” (RS cells), and these cells are characterized by two mirror image nuclei, each containing a large acidophilic they impart an “owl-eye” appearance.
- There are four recognized histologic types of Hodgkin’s disease.
- Lymphocyte predominant characterized by abundant lymphocytes, few plasma cells with occasional RS cells. This type carries the most favorable prognosis.
- Mixed cellularity characterized by lymphocytes, plasma cells, eosinophils and easily identifiable RS cells.
- Lymphocyte depletion this type shows sparse lymphocytes and stromal cells, with areas of fibrosis and highly malignant bizarre RS cells. This type carries the poorest prognosis.
- Nodular Sclerosis characterized by bands of collagen, subdividing the tumor cells into many small islands within the lymph node.

Treatment

By chemotherapy and radiotherapy. The overall prognosis is good and 5 years survival rate is about 80%.

MULTIPLE MYELOMA**Definition**

- Multiple myeloma represents a group of malignant tumors of bone, which develop from the bone marrow cells and are consisting of terminally differentiated B-lymphocytes or plasma cells.

Clinical Features

- Age → 40 to 70 years.
- Sex → Males are more frequently affected than females.
- Site → Bones anywhere in the skeleton can be affected and among

the jaw bones, mandible is more frequently affected than maxilla. Mandibular lesions are often multiple and they commonly occur over the molar – ramus region or the angle. In some cases, the lesion can occur in relation to the gingival tissue.

Clinical Presentation

- Severe bone pain is the most common early symptom of the disease with rapidly developing numbness of the lips or chin.
- The jaw lesions commonly produce fast enlarging, painful swelling, with expansion and destruction of the bone, which may even lead to the “egg-shell crackling” or pathological fractures. The regional teeth are usually mobile.
- Perforation of the cortex often causes protrusion of the lesion outside the bone as an ulcerated fleshy mass.
- Extraction of teeth in these patients usually causes severe hemorrhage.

Radiological Features

- In multiple myeloma, the radiograph shows numerous punched-out areas of radiolucency with no peripheral bone reaction. The lesions commonly involve the skull, vertebrae, ribs and jaw bones. Diffuse radiolucency in case of multiple myeloma may be seen occasionally.

Laboratory Investigations

- Most of the multiple myeloma patients exhibit hyperglobulinemia, with reversal of the serum albumin-globulin ratio.
- There will be an increase in the total serum protein level, measuring up to 8 to 16 gm%.
- Presence of Bence-Jones protein in urine is reported in most of the patients and it is one of the most important hallmarks in the diagnosis of the disease. This unusual protein coagulates when the urine is heated to the temperature of 42° to 60° C, it disappears when the urine is boiled and finally it reappears again as the urine is cooled. Occasionally the Bence-Jones protein in urine can also be present in patients with polycythemia or leukemia. However, the absence of this protein also does not rule out the presence of multiple myeloma.

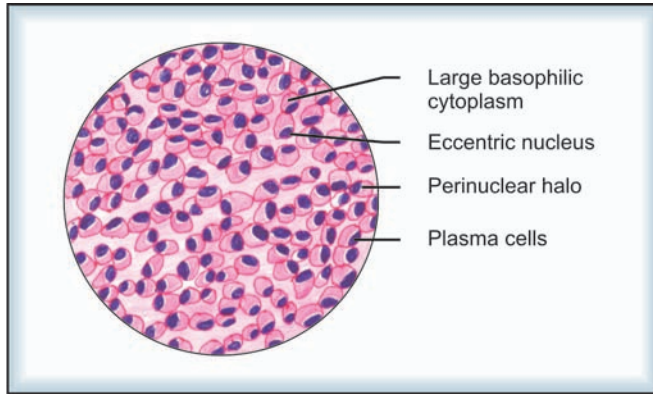


Fig. 12.28: Multiple myeloma

Histopathology

- The lesion is histologically characterized by sheets of closely packed, round or oval cells, resembling the plasma cells.
- These cells, with eccentrically placed nuclei often exhibit chromatin clumping in a “cart-wheel” or a “checkerboard” pattern.
- Mitotic figures may be high in few cases, with occasional presence of binucleated or multinucleated cells.

Treatment

- Chemotherapy is mostly given but the disease is fatal.

RHABDOMYOSARCOMA

- Rhabdomyosarcomas are malignant neoplasms of the striated muscle cells and they usually produce a non-descript, fast enlarging, soft tissue swelling in the tongue or in the palate of the young people.
- There are several types of rhabdomyosarcomas occurring in the oral cavity, namely the pleomorphic, embryonal, alveolar and botryoid type. Among these, the botryoid type is most commonly seen in the oral cavity. Microscopically this type of rhabdomyosarcoma presents numerous proliferating malignant rhabdomyoblast cells, in the form of multiple projecting lobulated mass,

resembling branch of grapes. The treatment is done by radical surgical excision followed by radiotherapy. Prognosis is poor.

NEUROGENIC SARCOMA

- Neurogenic sarcoma is a rare malignancy in the oral cavity, which develops either from the pre-existing lesions of neurofibromatosis or it may occur as a de novo lesion. The cell of origin of this tumor is probably the Schwann cells or the other nerve sheath cells.
- This soft tissue tumor usually produces a rapidly growing, painful, exophytic mass, which is otherwise a nondescript lesion. The bony lesion commonly involves the mandible (as the tumor often develops in relation to the mandibular nerve) and it produces either a small lesion causing dilatation of the mandibular canal or a large destructive swelling of the bone. Pain and paresthesia of the involved area are the most frequently encountered symptoms.
- Microscopically the neurogenic sarcoma consists of abundant spindle-shaped cells, with variable number of mitotic figures. The cells often show nuclear hyperchromatism with streaming and palisading nuclei.

METASTATIC TUMORS OF THE JAWS

- Metastatic tumors of the jaws are not very uncommon lesions and the primary tumors may be situated in a variety of locations in the body, e.g. bronchus, breast, kidney, colon, thyroid and prostate, etc.
- The metastatic tumor develops in the mandible four times as often as in the maxilla and the lesion mostly causes pain, swelling, paresthesia or anesthesia, loosening of teeth and pathological fractures, etc. In about 20 to 30 percent cases, these tumors are the first indication of the disease. Radiographs often reveal osteolytic or osteoblastic areas in the jaw and most of the tumors carry a grave prognosis.

Tumors of Salivary Gland

HISTOPATHOLOGICAL CLASSIFICATION OF SALIVARY GLAND TUMORS [WHO (1991)]

- These are a heterogeneous group of lesions of great morphologic variations and for this reason, present many difficulties in classification. These tumors are relatively uncommon. Eversole has proposed a histogenetic classification of salivary gland tumors, implicating two cell types as possible progenitors the intercalated duct cell and the excretory duct reserve cell. Salivary gland tumors are best distinguished by their histologic patterns. The clinical behaviour of these various lesions may be based, as in most tumors, on the type of tumor as well as on the method of treatment utilized.
- Tumors of salivary glands may arise not only from the major salivary glands, but also from any intraoral accessory salivary glands. Annual incidence of salivary gland tumors around the world is stated to be 1-6.5 cases per 1,00,000 people.
- Minor salivary gland tumors are more common in females than males, with a ratio range from 1.2 : 1-1.9:1. Benign tumors need to treatment other than surgical removal. Malignant tumors may require radiation chemotherapy or both after surgery.

- **Adenomas**
 - Pleomorphic adenoma
 - Myoepithelioma (Myoepithelial adenoma)
 - Basal cell adenoma
 - Warthin's tumor (Adenolymphoma)
 - Oncocytoma
 - Canalicular adenoma
 - Sebaceous adenoma
 - Ductal papilloma
 - Inverted ductal papilloma
 - Intraductal papilloma
 - Sialadenoma papilliferum.
- **Cystadenoma**
 - Papillary cystadenoma
 - Mucinous cystadenoma.
- **Carcinomas**
 - Mucoepidermoid carcinoma
 - Acinic cell carcinoma
 - Adenoid cystic carcinoma
(Cribriform/Tubular/Solid)
- Polymorphous low-grade adenocarcinoma (Terminal duct adenocarcinoma)
- Epithelial - Myoepithelial duct carcinoma
- Basal cell adenocarcinoma
- Sebaceous carcinoma
- Papillary cystadenocarcinoma
- Mucinous adenocarcinoma
- Oncocytic carcinoma
- Salivary duct carcinoma
- Adenocarcinoma
- Malignant myoepithelioma (Myoepithelial carcinoma)
- Carcinoma in pleomorphic adenoma (Malignant mixed tumor)
- Squamous cell carcinoma
- Small cell carcinoma
- Undifferentiated carcinoma
- Other carcinomas
- Nonepithelial tumors
- Malignant lymphomas
- Secondary tumors

- Unclassified tumors
- Tumor like disorders
- Sialadenosis (Siabosis)
- Oncocytosis
- Necrotizing sialometaplasia (Salivary gland infarction)
- Benign lymphoepithelial lesion
- Salivary gland cysts
- Chronic sclerosing sialadenitis of submandibular gland (Kuttner's tumor)
- HIV - related cyst and disorders.

CLASSIFICATION OF SALIVARY GLAND NEOPLASMS BY (ELLIS AND AUCLAIR, 1990)

(a) Primary Epithelial Neoplasms

Benign

- Mixed tumor (Pleomorphic adenoma)
- Papillary cyst adenoma lymphomatosum (Warthin's tumor)
- Oncocytoma
- Cystadenoma
- Basal cell adenoma
- Canalicular adenoma
- Ductal papillomas
- Sialadenoma papilliferum
- Inverted ductal papilloma
- Extraductal papilloma
- Myoepithelioma
- Sebaceous adenomas
- Sebaceous lymphadenoma
- Adenoma, not otherwise specified.

Malignant

- Low-grade mucoepidermoid carcinoma
- Low-grade acinic cell adenocarcinoma
- Polymorphous low-grade adenocarcinoma (terminal duct carcinoma).
- Basal cell adenocarcinoma

- Adeno carcinoma, not otherwise specified
- Intermediate grade
- Clear cell carcinoma
- Cystadenocarcinoma
- Papillary
- Non-papillary
- Sebaceous carcinoma
- Sebaceous lymphadenocarcinoma
- Mucinous adenocarcinoma.

High-Grade

- Mucoepidermoid carcinoma, high-grade
- Adenoid cystic carcinoma, solid
- Malignant and mixed tumor
- Carcinoma ex-mixed tumor
- Carcinosarcoma
- Adenocarcinoma, not otherwise specified, high-grade squamous cell carcinoma.
- Undifferentiated carcinoma
- Small cell carcinoma
- Lymphoepithelial carcinoma.

Others

- Oncocytic carcinoma
- Adeno squamous carcinoma
- Salivary duct carcinoma
- Myoepithelial carcinoma.

(b) Non-epithelial Neoplasms Major Salivary Gland

- Benign mesenchymal
- Hemangioma
- Schwannoma
- Neurofibroma
- Lipoma.

(c) Sarcomas

- Hemangiopericytoma
- Malignant schwannoma

- Fibrosarcoma
- Malignant fibrous histiocyoma
- Rhabdomyosarcoma.

(d) Others

- Lymphomas
- Non-Hodgkin's lymphoma
- Hodgkin's disease
- Metastatic neoplasms
- Malignant melanoma
- Squamous cell carcinoma
- Renal cell carcinoma
- Thyroid carcinoma.

TUMORS OF THE SALIVARY GLAND
BENIGN TUMORS OF THE SALIVARY GLANDS

Pleomorphic Adenoma

(Mixed tumor)

- It is the benign "mixed" tumor of salivary glands.
- Its morphologic complexity is the result of the differentiation of the tumor cells and the fibrous, hyalinized, myxoid, chondroid and osseous areas are the result of metaplasia or are products of tumor cells per se.
- It constitutes 90% of all benign salivary gland tumors.

Histogenesis

- These center around the myoepithelial cell and a reserve cell in the intercalated duct.
- Myoepithelial cell is responsible for the morphologic diversity of the tumor.
- Intercalated duct reserve cell can differentiate into ductal and myoepithelial cells and later can undergo mesenchymal metaplasia.
- Intercalated duct reserve cell is the histogenetic precursor of pleomorphic adenoma.

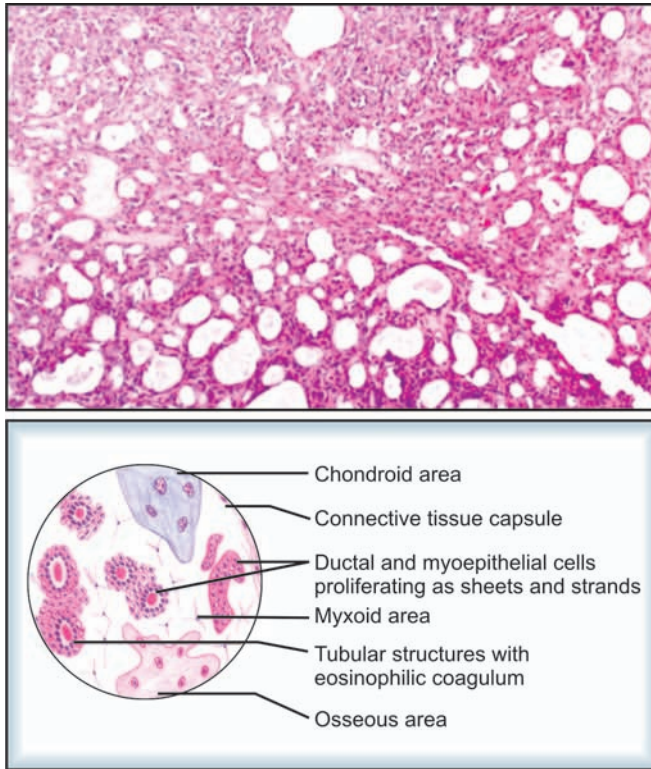


Fig. 13.1: Pleomorphic adenoma

- Neoplastically altered epithelial cell with the potential for multi-directional differentiation may be histogenetically responsible for pleomorphic adenoma.

Clinical Features

- 90% of the tumor occur in parotid gland.
- Females are commonly affected than males in the ratio female: male is 6:4.
- It is common in adults and children.
- It is small, painless, quiescent nodule which slowly begins to increase in size showing intermittent growth.
- It is irregular nodular lesion which is firm in consistency.
- Causes difficulties in mastication, talking and breathing.
- Palatal glands and glands of lip are frequently the site of origin.

Histologic Features

- Cuboidal cells are arranged in tubes or duct like structures which bear striking resemblance to normal ductal epithelium.
- Duct like spaces contain eosinophilic coagulum.
- There is often proliferation of epithelium in strands or sheets.
- In other areas, tumor cells assume stellate, polyhedral or spindle form.
- Hyaline cells (modified myoepithelial cells), squamous epithelial cells, keratin pearls, loose myxoid material, foci of hyalinized connective tissue, mucoid material are present.
- Tumor is encapsulated. When pleomorphic pattern of the stroma is absent and the tumor is highly cellular, it is often referred to as cellular adenoma.

Treatment

- Surgical excision.

PAPILLARY CYSTADENOMA LYMPHOMATOSUM

(Warthin's tumor, adenolymphoma)

- It occurs almost exclusively in the parotid gland, and occasionally in the submaxillary gland.

Histogenesis

- Tumor arises in salivary gland tissue entrapped within paraparotid or intraparotid lymph nodes during embryogenesis.
- It is most likely a delayed hypersensitivity disease, the lymphocytes being an immune reaction to the salivary ducts which undergo oncocytic change.
- The lymphoid component of the tumor is an exaggerated secretory immune response.

Clinical Features

- Males are more commonly affected. Age 41 to 70 years.
- Tumors are bilateral.
- Tumor is generally superficial, lying just beneath the parotid capsule.
- It is a painless lesion and firm on palpation.

Histologic Features

- It is made up of two histologic components
 1. Epithelial
 2. Lymphoid tissue.
- The lesion is essentially an adenoma exhibiting cyst formation with papillary projections into the cystic spaces and a lymphoid matrix showing germinal centers.
- The epithelial cells covering the papillary projections are columnar and cuboidal cells usually arranged in 2 rows.
- The cells are eosinophilic, containing hyperchromatic or pyknotic nuclei and vast numbers of mitochondria.
- There is an eosinophilic coagulum present within cystic spaces which appears as chocolate color fluid macroscopically.

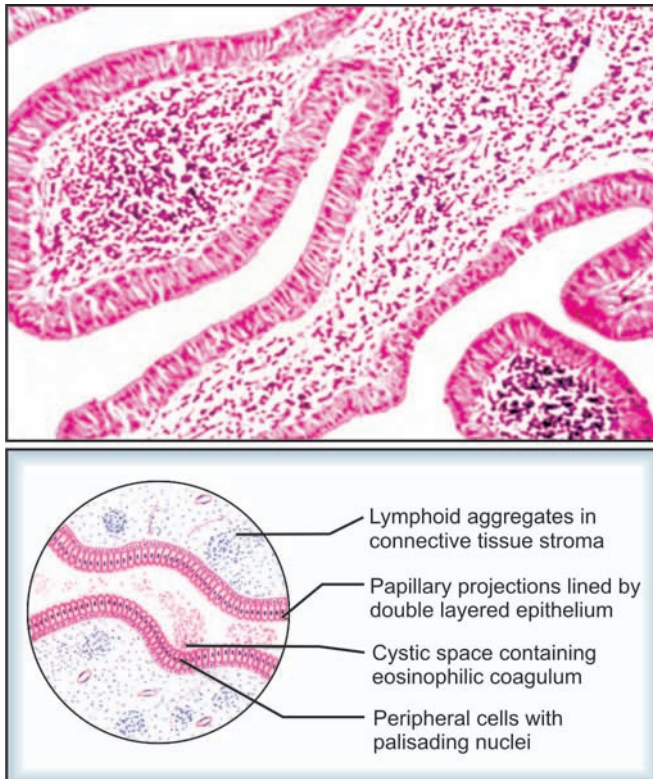


Fig. 13.2: Warthin's tumor

- Abundant lymphoid component may represent the normal lymphoid tissue or cellular infiltrate which involves both humoral and cell mediated mechanism.

Treatment

- Surgical Excision.

OXYPHILIC ADENOMA

(Oncocytoma, acidophilic adenoma)

- It usually occurs in the parotid gland.
- "Oncocytoma" is derived from the resemblance of these tumor cells to apparently normal cells which have been termed "Oncocytes" and which are found in a great number of locations, including the salivary glands, respiratory tract, breast, thyroid, pancreas, parathyroid, pituitary, testicle, fallopian tube, liver and stomach.

Clinical Features

- It is more common in women.
- It occurs in elderly persons, mostly between the age of 51 to 80 years.
- Tumor measures 3-5 cm in diameter, appears as discrete, encapsulated mass, sometimes nodular.

Histologic Features

- There are large cells which have eosinophilic cytoplasm and distinct cell membrane, which tend to be arranged in narrow rows or cords.
- Cells occur in sheets and may demonstrate alveolar or lobular pattern.
- They exhibit few mitotic figures, are closely packed and there is little supportive stroma.
- Lymphoid tissue is frequently present.
- Cells are engorged with enlarged and morphologically altered mitochondria.

Treatment

- Surgical excision.
- In the parotid gland, partial parotidectomy.
- In submandibular gland, total removal of gland.

MYOEPIITHELIOMA

- It is an uncommon salivary gland tumor which accounts for less than one percent.

Clinical Features

- Occurs in adults with equal sex distribution. Parotid gland is most commonly involved. Palate is most frequent intraoral site.

Histologic Features

- Composed of spindle shaped or plasmacytoid cells or a combination of 2 cell types. Definite diagnosis lies in the ultra-structural identification of myoepithelial cells. Myoepithelial cell exhibits basal lamina and fine intracytoplasmic myofilaments.
- Desmosomes are encountered between adjacent cells.

Treatment

- Surgical excision.

BENIGN LYMPHOEPITHELIAL LESION

(Mikulicz disease)

- It exhibits both inflammatory and neoplastic characteristics.
- There is increasing evidence that the disease is closely related to Sjögren's syndrome and is an autoimmune disease in which the patients own salivary gland tissue becomes antigenic.

Clinical Features

- Develops in adults with a mean age of 50 years. 60 to 80 percent cases occur in women. 85 percent cases occur in parotid gland.
- It usually appears as a firm, diffuse swelling of the affected gland.
- It may be asymptomatic as associated with mild pain and Xerostomia.

- Manifested essentially as unilateral /bilateral enlargement.
- Onset of lesion is with fever, upper respiratory tract infection, oral infection, tooth extraction.
- Lacrimal glands are also enlarged.

Histologic Features

- There is heavy lymphocytic infiltrate associated with destruction of salivary acini.
- Ductal epithelium persist.
- Ductal cells and myoepithelial cells become hyperplastic, forming highly characteristic groups of cells known as epimyoeplithelial islands throughout the lymphoid proliferation.

Treatment

- Surgical removal of the involved gland have an increased risk for lymphoma.

Sjögren's Syndrome

- Is a chronic systemic autoimmune disorder that principally involves the salivary and lacrimal glands resulting in xerostomia and keratoconjunctivitis and rheumatoid arthritis.

Two Forms of Disease

- Primary Sjögren's syndrome –xerostomia, keratoconjunctivitis (sicca complex)
- Secondary Sjögren's syndrome –xerostomia, keratoconjunctivitis rheumatoid arthritis, lupus erythematosus, polyarteritis nodosa, polymyositis or scleroderma.

Etiology

- There is evidence of genetic influence.
- Histocompatibility antigens (HLA) are found with greater frequency.

- HLA - DRW 52 is associated with both forms of disease.
- HLA - B8 and HLA DR3 are in primary form.
- San Diego criteria for Sjögren's syndrome.

Clinical and Radiographic Features

- Prevalence of syndrome is 0.2 to 3.0%.
- 80 to 90 percent cases occur in females 10:1.
- Predominantly in middle aged adults.
- When associated with another connective tissue disease it is called secondary Sjögren's syndrome.
- It is commonly associated with rheumatoid arthritis.
- Secondary Sjögren's syndrome may develop in 30 percent of patients with SLE.
- Principal oral symptom is xerostomia.
- Saliva may appear frothy, with a lack of the usual pooling saliva in the floor of mouth.
- Patients may have difficulty in swallowing, altered taste or difficulty in wearing dentures.
- Tongue becomes fissured and exhibits atrophy of the papillae.
- Oral mucosa may be red and tender as a result of secondary candidiasis
- Denture sore mouth and angular cheilitis are common.
- Lack of salivary cleansing action predisposes the patient to dental decay especially cervical caries.
- 1/3rd to 1/2 of patients have diffuse, firm enlargement of major salivary gland.
- Swelling is bilateral, nonpainful, tender, intermittent or persistent in nature.
- Reduced salivary flow places these individuals at increased risk of retrograde bacterial sialadenitis.
- Sialographic examination often reveals punctuate sialectasia and lack of normal association of ductal system demonstrating "branchless-fruit-laden tree" pattern or "cherry-blossom pattern".
- Primary Sjögren's syndrome.
- Symptoms and objective signs of ocular dryness.
- Schirmer's test.
- Positive Rose Bengal Staining of cornea or conjunctiva to demonstrate *Keratoconjunctivitis sicca*.

- Symptoms and objective signs of dry mouth.
- Decreased parotid flow rate using lashley cups or other methods.
- Abnormal findings from biopsy of minor salivary gland.
- Serologic evidence of a systemic autoimmunity.
- Elevated rheumatoid factor > 1:320.
- Elevated antinuclear antibody > 1:320.
- Presence of anti SS-A(Ro) or anti SS-B (Ca) antibodies.
Biopsy of minor salivary glands of lower lip is taken. Finding of more than one focus of 50 or more lymphocytes and plasma cells within a 4 mm² area of glandular tissue is considered diagnosis of Sjögren's syndrome.
- Scintigraphy with radioactive technetium - 99 in pertechnetate shows decreased uptake and delayed emptying of the isotope.
- Sjögren's syndrome is a systemic disease and inflammatory process can affect other body tissues.
- Skin is dry, as are the nasal and vaginal mucosa.
- Fatigue is common, depression can occur.
- Other problems include lymphadenopathy, primary biliary cirrhosis, Raynaud's phenomenon, interstitial nephritis, myositis, vasculitis and peripheral neuropathies.

Lab Values

- **ESR rate is high.**
- Polyclonal hyperglobulinemia.
- Variety of autoantibodies are produced.
- Positive rheumatoid factor is found in 75% cases.
- Antinuclear antibodies are also present especially anti SS-A and anti SS-B in primary Sjögren's syndrome.
- Anti salivary duct autoantibodies can be demonstrated in secondary Sjögren's syndrome.
- Salivary protein electrophoresis is an useful test for diagnosing Sjögren's syndrome.

Histologic Features

- a. Lymphocytic infiltration of salivary glands with destruction of acinar units.
- b. Major glands shows progression to a lymphoepithelial lesion with characteristic epimyoepithelial islands.
- c. Atrophy of the glands.

Treatment

- It is mostly supportive.
- Dry eyes are best managed by use of artificial tears.
- Sealing the lacrimal puncture at the inner margin of eyelids can be helpful by blocking the normal drainage of lacrimal secretions into nose.
- Artificial saliva substitutes like sugarless candy or gum can help to keep the mouth moist.
- Symptoms can also be relieved by use of lactoperoxidase, lysozyme and lactoferrin.
- Sialogogues such as pilocarpine are used to stimulate salivary flow.
- Because of increased risk of dental caries daily fluoride applications may be indicated.
- Antifungal therapy is often needed to treat secondary candidiasis.

MALIGNANT TUMORS OF SALIVARY GLANDS

MALIGNANT PLEOMORPHIC ADENOMA

(Carcinoma ex-pleomorphic adenoma)

- Represent malignant counterparts to the benign mixed tumor or pleomorphic adenoma.
- Characterized by malignant transformation of the epithelial component of a previously benign pleomorphic adenoma.

Clinical Features

- Most common in middle aged and older adults with a peak prevalence in the 6th to 8th decade.
- Patient may report that a mass has been present for many years undergoing a recent rapid growth with associated pain or ulceration.
- 80% of cases are seen within major glands primarily the parotid.
- There is slight female predilection.
- May present as a painless mass also.
- Parotid tumors produce facial nerve palsy.

Histologic Features

- There are areas of malignant degeneration of the epithelial component characterized by cellular pleomorphism and abnormal mitotic activity.
- It may often have an aggressive growth pattern, with capsular invasion and infiltration into surrounding tissues.

Treatment

- Treatment done by wide excision possibly in conjunction with local lymph node dissection and adjunctive radiation therapy.
- Overall 5 year survival rate ranges from 25 to 65 percent but this rate drops 10 to 35 percent at 15 years.
- Prognosis is dependent on histopathologic subtype of the malignant component.

MUCOEPIDERMOID CARCINOMA

- It is one of the most common salivary gland malignancies.

Clinical Features

- It makes up to 10 percent of all major salivary gland tumors and 15 to 21 percent of minor gland tumors.
- It occurs between 2nd to 7th decades of life.
- It is the most common malignant salivary gland tumor in children.
- There is slight female predilection.
- It is most common in parotid gland and appears as asymptomatic swelling.
- Pain or facial nerve palsy may develop in association with high grade tumors.
- Minor glands constitute the second most common site.
- It is found in lower lip, floor of mouth, tongue and retromolar pad area.

Histologic Features

- The mucoepidermoid carcinoma is composed of a mixture of mucous producing cells and squamous cells.
- Mucus cells vary in shape but contain abundant foamy

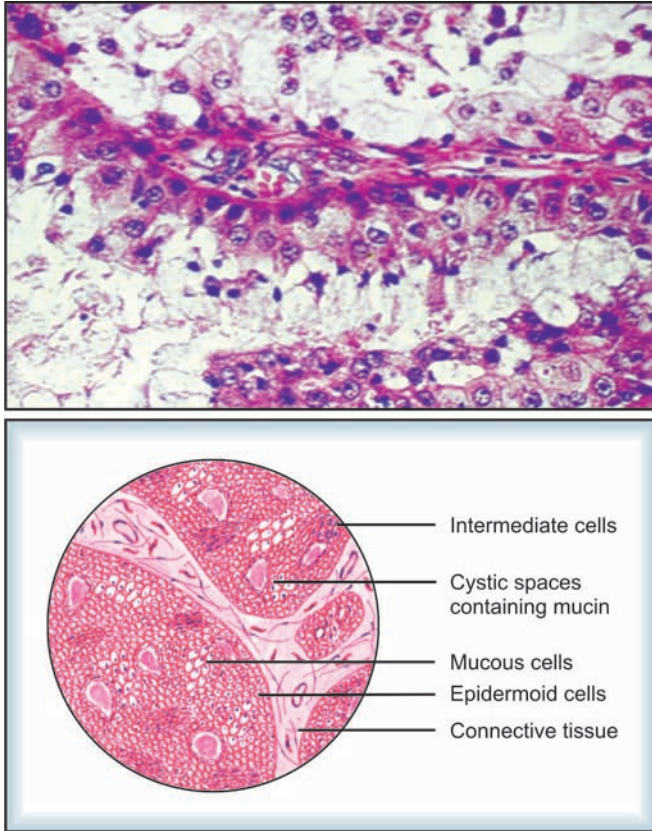


Fig. 13.3: Mucoepidermoid carcinoma

cytoplasm that stains positively with mucin stains.

- Epidermoid cells are characterized by squamoid features often demonstrating a polygonal shape, intercellular bridges, rarely keratinization.
- A third type of cells is typically present the intermediate cell.
- The cell is basaloid in appearance and is believed to be a progenitor of the mucous and epidermoid cells.
- Some tumors also show variable numbers of clear cells.
- An associated lymphoid infiltrate is present.
- Mucoepidermoid carcinoma have been categorized into one of 3 histopathologic grades based on the following:
 - a. Amount of cyst formation.

- b. Degree of cystologic atypia.
- c. Relative number of mucous, epidermoid and intermediate cells.

(a) Low-grade

- Tumors show prominent cyst formation, minimal cellular atypia and a relatively high proportion of mucous cells.

(b) High-grade

- Tumors consist of solid islands of squamous and intermediate cells which can demonstrate considerable pleomorphism and mitotic activity.

(c) Intermediate-grade

- Show features that fall between those of low-grade and high-grade neoplasms. Intermediate cells usually predominate.

Treatment

- In early stage tumors, subtotal parotidectomy with preservation of the facial nerve.
- In advanced tumors, total removal of parotid gland with sacrifice of facial nerve.
- Radical neck dissection is indicated for patients with clinical evidence of metastatic disease.
- Postoperative radiation therapy may be used for more aggressive tumors.

ACINIC CELL ADENOCARCINOMA

- Is a salivary gland malignancy with cells that show serous acinar differentiation.

Clinical Features

- 85 percent of tumor occur in the parotid gland.

Site

- Buccal mucosa, lips, palate are commonly affected.

- About 8.6 percent of all parotid tumors, 2.7 to 4 percent of submandibular gland tumors, 9 percent of oral minor salivary gland tumors constitute Acinic Cell Adenocarcinoma.

Age

- It occurs between second to seventh decade, 60 percent cases occur in women.
- It is a growing mass, tumor may be asymptomatic with associated pain or tenderness.

Histologic Features

- Tumor is well circumscribed, appear encapsulated, exhibiting an infiltrative growth pattern.
- Most characteristic cell is one with features of serous acinar cell, with abundant granular basophilic cytoplasm and a round, darkly stained eccentric nucleus.
- Several growth patterns are seen.

(a) Solid

- Solid variety consists of numerous well differentiated acinar cells arranged in a pattern that resembles normal parotid gland tissue.

(b) Microcystic

- Multiple small cystic spaces are created that contain some mucinous or eosinophilic material.

(c) Papillary Cystic

- Larger cystic areas are formed that are lined by epithelium having papillary projections into the cystic spaces.

(d) Follicular

- Has an appearance similar to that of thyroid tissue.

Treatment

- If limited to the superficial lobe of parotid gland, lobectomy.
- For those in deep lobe, parotidectomy.

- Facial nerve may be sacrificed if it involves the tumor.
- Submandibular tumors are managed by total removal of the gland.
- Active radiation therapy may be considered for uncontrolled disease.
- 1/3rd of patients have recurrence.
- Metastasis develop in 10 to 15 percent of patients.

ADENOID CYSTIC CARCINOMA

(*Cylindroma*)

- It is one of the more common and best recognized salivary malignancies.

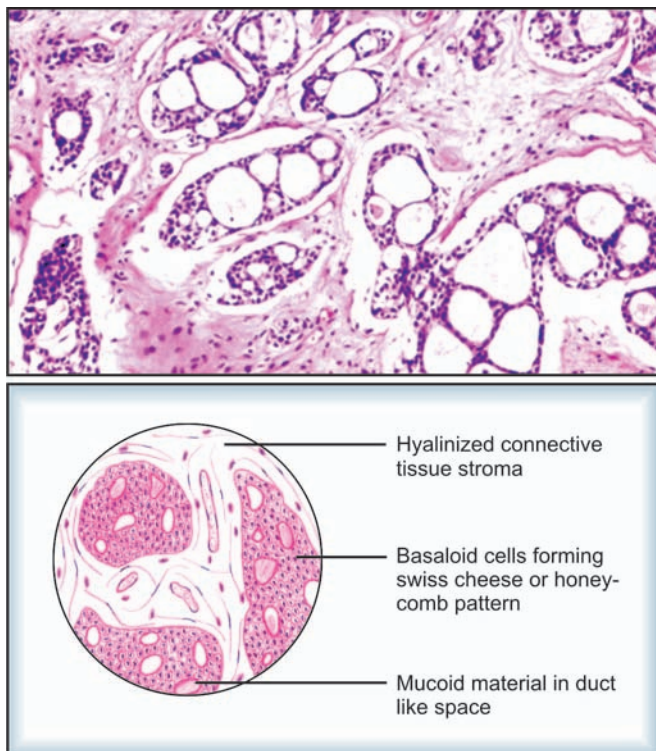


Fig. 13.4: Adenoid cystic carcinoma

Clinical Features

- 50 percent of cases develop in minor salivary glands.
- Palate is the most common site.
- Parotid and submandibular gland are also involved.
- Most common in middle aged adults.
- Appears as a slowly growing mass.
- Pain is a common finding.
- Patients complain of a constant, low-grade, dull ache which gradually increases in intensity.
- Facial nerve paralysis may develop.

Histologic Features

- It is composed of a mixture of myoepithelial cells and ductal cells.
- Three major patterns are recognized
 1. Cribriform
 2. Tubular
 3. Solid.

1. Cribriform Pattern

It is characterized by islands of basaloid epithelial cells that contain multiple cyst like space or resembling swiss cheese or honeycomb pattern.

- These spaces often contain mildly basophilic material, hyalinized eosinophilic product or a combined mucoid hyalinized appearance.
- Small strands of tumor are found embedded within this hyalinized stroma.
- Tumor cells are small, cuboidal exhibiting deeply basophilic nuclei and little cytoplasm.

2. Tubular Pattern

- Occurs as multiple small tubules within a hyalinized stroma.

3. Solid Pattern

- Both ductal cells and myoepithelial cells are present as nests with focal necrosis in the center of tumor islands.

- There is tendency to show perineural invasion which corresponds to a pain.

Treatment

- Surgical excision and adjuvant radiation therapy.
- Tumor with solid histopathologic pattern are worst affected.

NECROTIZING SIALOMETAPLASIA

- Necrotizing sialometaplasia is an uncommon destructive inflammatory condition of the salivary glands.
- It is the result of ischemia of salivary tissue that leads to local infarction.

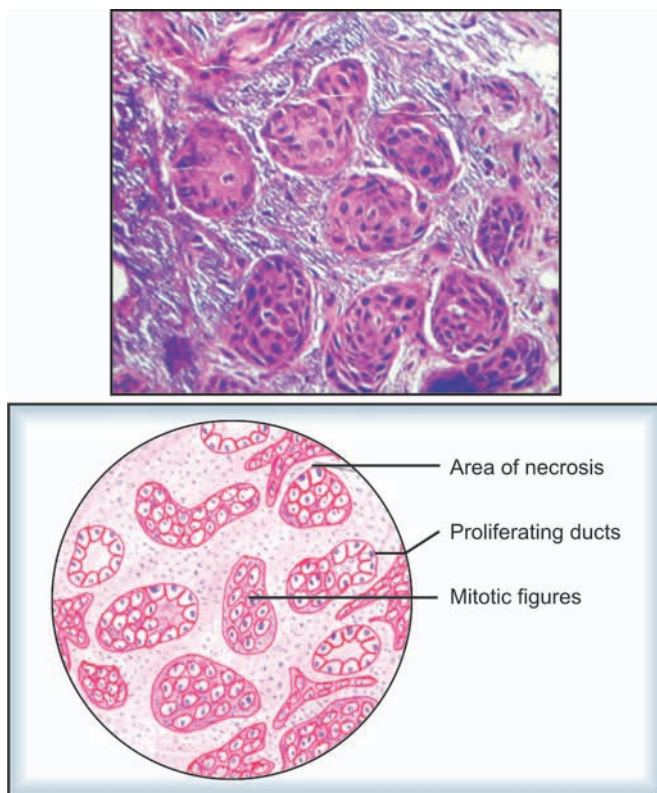


Fig. 13.5: Necrotizing sialometaplasia

- Potential predisposing factors are:
 - Traumatic injuries.
 - Dental infections.
 - Ill fitting dentures.
 - Upper respiratory tract infections.
 - Adjacent tumors.
 - Previous surgery.

Clinical Features

- Most frequently develops in palatal salivary glands. 75% cases occur on posterior palate.
- 2/3rd of cases are unilateral.
- Most common in adults, mean age of onset is 46 years. Males are affected twice as often as females.
- Appears as a nonulcerated swelling associated with pain and paresthesia.
- Within 2-3 weeks, necrotic tissue sloughs out, leaving a crater like ulcer that can range from less than 1cm to more than 5 cm.

Histologic Features

Characterized by acinar necrosis in early lesions followed by associated squamous metaplasia of the salivary ducts.

- Mucous acinar cells are necrotic
- There may be liberation of mucin with an associated inflammatory response.

Treatment

- Biopsy is usually indicated.
- No specific treatment is indicated.
- Lesion resolves on its own with an average healing time of 5 to 6 weeks

CLEAR CELL CARCINOMA

Histogenesis

- Semipluripotential intercalated duct cell has been implicated.
- Even myoepithelial cell is the progenitor of the clear cell.

Clinical Features

- Found primarily in the major salivary glands especially parotid.
- Occurs in elderly adults, more often in females.

Histologic Features

- Are composed of clusters of cells surrounded by thin septa of fibrous connective tissue.
- Produces an “organoid” appearance.
- It consists of an inner layer of ductal cells.
- Surrounded by a layer of clear cells.
- glycogen may be demonstrated in the cells by PAS reaction.

Treatment

- Treated by surgical excision.
- It has a favorable prognosis and less than 1/3rd recur and few metastasize.

Cysts and Tumors of Oral Cavity

Definition

According to Kramer, cyst is defined as a pathologic cavity having fluid, semifluid, or gaseous content which is not created by accumulation of pus and is frequently but not always lined by epithelium.

- Cyst and tumors of odontogenic origin constitute an unusually diverse group of lesions. Normal odontogenesis is a multistep complex process. Aberration in this process leads to development of these lesions. An understanding of the pathogenesis of the odontogenic tumors is predicted upon an understanding of the histogenesis of the tooth.
- The odontogenic cysts are included here because they too represent an aberration at some stage of odontogenesis and somewhere they are intimately associated with the development of certain odontogenic tumors. The current classification is based upon the origin from various germ layers.

Classification

CYSTS OF JAWS	
<i>Epithelial</i>	<i>Non-epithelial</i>
a. Odontogenic	- Traumatic bone cyst
• <i>Developmental</i>	- Solitary bone cyst
- Gingival cysts of infants	
- Odontogenic keratocyst	
- Dentigerous cyst	
- Lateral periodontal cyst and median periodontal cyst	
- Gingival cyst of adults mandibular cyst	
- Glandular odontogenic cyst	
- Calcifying epithelial odontogenic cyst	
• <i>Inflammatory</i>	
- Radicular cyst	
- Residual cyst	
- Paradental cyst	
b. Non odontogenic	
- Nasopalatine cyst	
- Nasolabial cyst	
- Globulomaxillary cyst	
- Median palatine	

DENTIGEROUS CYST

- It is a developmental odontogenic cyst that surrounds the crown of an impacted tooth.
- It is caused by the accumulation of fluid between the reduced enamel epithelium and enamel surface.
- This leads to the crown being situated within the cystic lumen.
- One of the commonest types of developmental odontogenic cyst.
- Seen in 20 percent of all jaw cyst.

Clinical Features

- Associated with crown of an impacted, embedded or unerupted tooth.
- Also seen associated with supernumerary tooth.
- Most common site → Mandibular and maxillary third molar, maxillary cuspid area.
- Lesions seen in second and third decade.
- Slight predilection to males.
- Most cysts are solitary and unilateral.
- Bilateral and multiple lesions are associated with cleidocranial dysplasia and Maroteaux-Lamy syndrome.
- The lesion may become aggressive.

- The cystic enlargement causes bone expansion, facial asymmetry, displacement of tooth, root resorption and pain.
- Usually patients will be asymptomatic until the cyst becomes secondarily infected.

Radiological Features

- Radiolucent area with an unerupted tooth crown.
- Normal follicular space is usually 3-4 mm and the dentigerous cyst can be suspected if the follicular space is more than 3 mm.
- Radiologically lesion shows three types or varieties.
Central: The crown is enveloped symmetrically.
Lateral: Dilatation of follicle on one side of tooth crown.
Circumferential: Follicle expands in such a manner that the entire tooth appears to be enveloped by the cyst.
- Some times the radiolucent area is surrounded by a thin sclerotic border representing bone reaction.

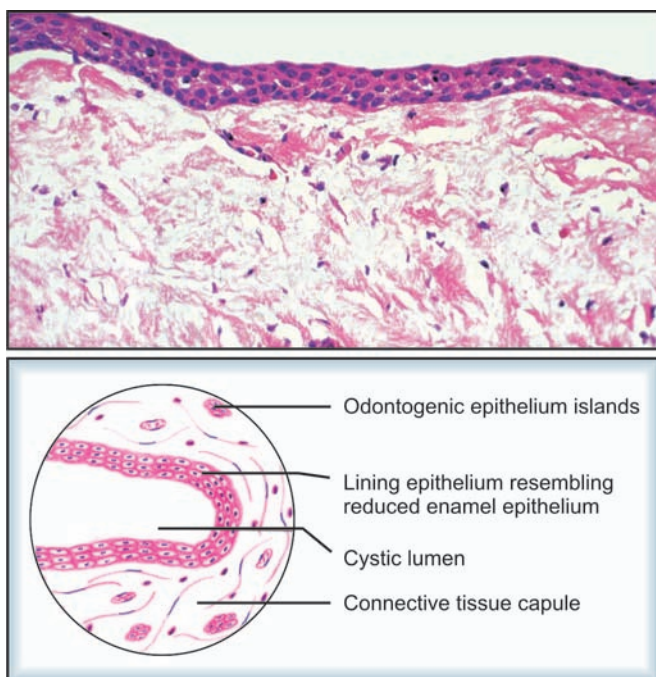


Fig. 14.1: Dentigerous cyst

Histologic Features

- Composed of thin layer of epithelium and connective tissue.
- Epithelium is of stratified squamous type lining the lumen.
- Retepeg formation is absent.
- The connective tissue wall is frequently quite thickened and composed of loose fibrous connective tissue.
- Connective tissue shows varying numbers of islands of epithelium.
- Later stages it may transform into ameloblastoma.
- Inflammatory cell infiltrate is quite evident.
- Inflammatory dentigerous cysts exhibits Rushton bodies.

Formation of Cyst

- Formed by reduced enamel epithelium. Formation occurs in following stages.
- The increased hydrostatic pressure separates the follicle from crown with or without reduced enamel epithelium.
- With advent of time, the capillary permeability is increased so as to permit the passage of greater quantities of proteins above the low concentration of pure transudate.
- Thus a cystic wall is formed.
- The wall contains glycosaminoglycans.
- This gets diffused into the cystic fluid.
- This helps in expansile growth by increasing osmolality of cystic fluid, hence raising the hydrostatic pressure of cyst.

Treatment

- Depends upon size of the lesion. Smaller cysts can be treated by enucleation and larger cysts are removed by marsupialization.

Complications

- Development of ameloblastoma.
- Development of epidermoid carcinoma.
- Development of mucoepidermoid carcinoma.

ODONTOGENIC KERATOCYST

- Developmental odontogenic cyst.
- Derived from remnants of dental lamina.
- Shows biologic behavior similar to benign neoplasms.
- OKC was first coined by Philipsen in 1956.
- In 1963 Pindborg and Hansen described essential features of cyst.
- The cyst is named so because of Keratin that fills the cystic lumen.
- They are aggressive lesions and associated with nevoid basal cell carcinoma syndrome.
- In 1967 Toller regarded OKC as benign neoplasm.
- Orthokeratinized odontogenic cyst shows less aggressive behaviour.

Formation of Cyst

- OKC arises from odontogenic epithelium which is derived from dental lamina or its remnants and extension of basal cells from overlying epithelium.
- The stimulus to OKC is genetically determined.

Clinical Features

- Occurs at any age, especially in younger adults.
- Males are commonly affected.
- Mandible is affected commonly than maxilla.
- In mandible, ramus, third molar area is commonly involved.
- In maxilla common site is third molar region.
- Patients exhibit pain, soft tissue swelling and bone expansion and neurologic manifestations such as paresthesia of lower lip which is common.

Radiological Features

- Most odontogenic keratocyst are unilocular with well defined peripheral rim of sclerotic bone.
- Multilocular radiolucent lesion are also seen with central cavity having satellite cysts.

Radiologically, it is seen as:

- *Replacement type*: Occur in place of normal tooth.
- *Envelopmental type*: Surrounds the crown.

Histologic Features

- Shows epithelial and connective tissue parts.
- Epithelium is stratified squamous type.
- Shows parakeratinization with corrugation.
- Thickness of epithelium ranging from 6 to 10 cells thick.
- Palisaded, polarized basal layer of cells often described as having “picket fence” or “tombstone” appearance.
- Connective tissue shows small islands of epithelium similar to the lining epithelium.
- Some of these islands may be small cysts.
- Lumen is filled with Keratin and straw colored fluid with thicker creamy material.
- At the sites of inflammation cholesterol clefts and hyaline bodies are present.
- Parakeratinized cysts show increased tendency for recurrence.

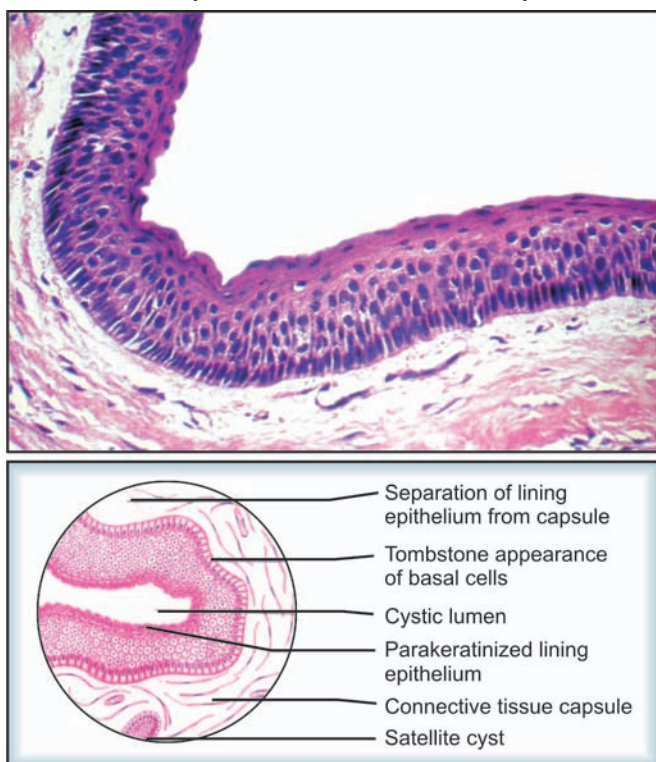


Fig. 14.2: Odontogenic keratocyst

Treatment

- By following methods.
 - Marsupialization.
 - Enucleation and primary closure.
 - Enucleation and packing open.

Causes for Recurrence

- Presence of the fragile epithelium.
- Presence of daughter cysts.

JAW CYST BASAL CELL NEVUS BIFID RIB SYNDROME

(Basal Cell Nevus Syndrome, Gorlin and Goltz syndrome)

- This syndrome was first described by Binkley and Johnson in 1951.
- Later on it has been thoroughly reviewed by Gorlin and his workers.
- It is a hereditary condition, transmitted as an autosomal dominant trait, with high penetrance.

Clinical Features

- The syndrome is very complex and includes a great variety of possible abnormalities, as follows
- Cutaneous anomalies, e.g. basal cell carcinoma, benign dermal cysts and tumors, palmar pitting, palmar and plantar keratosis and dermal calcinosis.
- Dental and osseous anomalies → OKC (multiple) mild mandibular prognathism, bifid rib, vertebral anomalies and brachy metacarpalism.
- Ophthalmologic anomalies → Hypertelorism with wide nasal bridge, dystopia canthorum, congenital blindness and internal strabismus.
- Neurologic anomalies → Mental retardation, dural calcification, agenesis of corpus callosum, congenital hydrocephalus and occurrence of medulloblastomas with greater than normal frequency.
- Sexual abnormalities → Hypogonadism in males and ovarian tumors in females.

- Some patients show lack of response to parathormone by the lack of phosphate diuresis which, coupled with shortened fourth metacarpals.

DENTAL LAMINA CYSTS OF NEWBORN

(Gingival cyst of newborn, Epstein pearls, Bohn's nodules)

- They are usually multiple but occasionally solitary lesions.
- Show superficial nodules on edentulous alveolar ridges of infants.
- They resolve without any treatment.
- They are derived from the rests of dental lamina.
- They contain keratin producing epithelial lining.
- Bohn's nodules and gingival Epstein pearls are similar lesions.
- Usually they are confused with gingival cyst but differentiated by location and etiology.
- Epstein pearls are located along the mid palatine raphae.
- They are derived from entrapped epithelial remnants along line of fusion.
- Bohn's nodules are scattered along the palate and seen most numerous at the fusion of hard and soft palate.
- They are derived from the palatal salivary gland structures.

Clinical Features

- Occasionally they become enlarged and present as discrete white swellings of alveolar ridge.
- They are usually asymptomatic.

Histologic Features

- They are true cysts.
- Show epithelial lining.
- They lack rete processes.
- Lumen is usually filled with desquamated Keratin.
- Sometimes dystrophic calcification and hyaline bodies of Rushton are also seen.

GINGIVAL CYST OF ADULTS

- Odontogenic cyst of gingiva.
- Derived from cell rests of dental lamina.

- They contain lining of embryonic epithelium.
- Occurs in free or attached gingiva.

Etiopathogenesis

- Heterotropic glandular tissue.
- Degenerative changes in proliferative epithelium.
- Traumatic implantation of epithelium.
- Remnants of dental lamina.
- There are two forms:
 1. That arising from cystic transformation of dental lamina or glands or rests of Serre.
 2. That arising from traumatic implantation of surface epithelium.
- This is an extraosseous counterpart of lateral periodontal cyst.

Clinical Features

- Occurs at any age but most common in adult.
- Mean age of occurrence is 40 years.
- Location -> Mandibular bicuspid, cuspid and incisor region.
- Present as a small, well circumscribed painless swelling of gingiva.
- Sometimes resembles mucocele.
- Lesion is of some color compared with adjacent normal tissue.

Histologic Features

- Epithelium thickness ranges from single cell layer to several layers thick.
- Glycogen rich clear cells may be present.
- They may be unicystic or polycystic.
- Sometimes calcification may be seen.

Treatment

- Surgical excision.

LATERAL PERIODONTAL CYST

- Developmental odontogenic cyst.
- Derived from one or more dental lamina remnants.
- Located in lateral periodontal location.

- Arises from cystic degeneration of clear cells of dental lamina.
- They are in intimate association with lateral root surface of any erupted tooth.
- Commonly seen in mandibular bicuspid area.
- The cyst may originate from dentigerous cyst.
- Proliferation of cell rests of malassez in the periodontal ligament.
- Arises from proliferation and cystic transformation of rests of dental lamina.
- An unusual form of cyst termed botryoid odontogenic cyst which showed multilocular pattern radiologically and was histologically and clinically similar to lateral periodontal cyst.
- Now, it is named as polycystic variant of lateral periodontal cyst.

Clinical Features

- Occurs in adults.
- Mean age is 50 years.
- Males > Females.
- Most patients were asymptomatic.

Radiologic Features

- Radiolucent lesion at the lateral surface of tooth root.
- Lesion seldom shows more than 1cm in diameter.

Histologic Features

- Shows, thin nonkeratinized epithelium.
- Usually one to five cells thick.
- Usually resembles reduced enamel epithelium.
- Cuboidal/columnar cells may be found.
- Many cells shows glycogen rich clear cells.
- Connective tissue exhibits zone of hyalinization.

Treatment

- Surgical excision.

CALCIFYING ODONTOGENIC CYST

(Gorlin Cyst)

- Cystic keratinizing tumor.
- This cyst is derived from odontogenic epithelium.

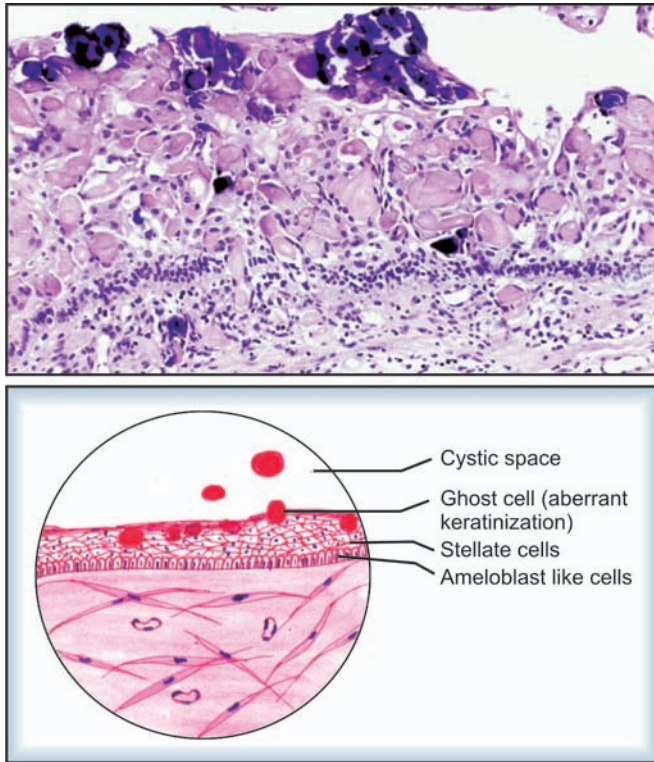


Fig. 14.3: Calcifying epithelium odontogenic cyst

- Resembles variety of clinicopathologic and behavioral features.
- Because of this diversity the 1992 WHO classification includes this cyst and all its variants in the category of odontogenic tumor.
- It exhibits two lesions;
 - Cystic lesion
 - Solid neoplastic lesion.
- Present as an intraosseous cystic lesion.
- Sometimes represents as peripheral lesion.
- The neoplasm is termed as "dentiginous ghost cell tumor".

GLANDULAR ODONTOGENIC CYST

(*Sialo odontogenic cyst, Mucoepidermoid odontogenic cyst*)

- Solitary/multiple odontogenic cyst.
- Derived from the rest of dental lamina.
- Epithelium contains numerous mucous secreting cells.

Clinical Features

- Occurs at wide age range.
- Male > Females.
- Anterior mandible is commonly involved.
- Lesions show slow progression.
- They are locally destructive and painless.

Radiological Features

- Will defined with multilocular radiolucency.

Histologic Features

- Lined by non-keratinized epithelium.
- Epithelium shows (goblet) mucous producing cells.
- The microcysts open and gives papillary or corrugated appearance.

Treatment

- Surgical removal.

RADICULAR CYST

(Periapical cyst, Apical periodontal cyst, Root end cyst)

- Most common odontogenic inflammatory cyst.

Etiology

- Infection of tooth leads to necrosis of pulp. Toxin releases through the apex and stimulates the inflammation and the epithelial rests of malassez, which is found in apical periodontal ligament, resulting in periapical granuloma.
- Eventually, the epithelium undergoes necrosis caused by lack of blood supply.
- Granuloma becomes cyst.

Clinical Features

- They occur in any tooth in the periapical area.
- They occur at any age.
- Seldom seen associated with primary dentition.
- Usually asymptomatic.

- Causes destruction of bone.
- Causes expansion of cortical plates.

Radiological Features

- Periapical round or oval radiolucency of variable size surrounded by a radiopaque margin.

Histologic Features

- It is limited by a mature collagenous connective tissue wall.
- Abundant fibroblasts can be seen in the cystic wall with inflammatory infiltrate.
- Lymphocytes are the most common cells present.
- They show darkly stained nucleus.
- Long standing lesions show plasma cells (Elongated cells with cart wheel shaped nucleus).
- Occasionally areas of hemorrhage are also seen.
- Multinucleated giant cells are also seen.
- Epithelium is stratified squamous epithelium.
- They are lined by respiratory epithelium if the lesions are in the vicinity of maxillary sinus.
- More number of Rushton bodies and hyaline bodies are seen.

Treatment

- Extraction of involved tooth and careful curettage, some conditions, root canal therapy with apicectomy is done.

RESIDUAL CYST

- They are retained periapical cysts from teeth that have been removed.
- This will develop in a dental granuloma that is left after an extraction.
- May be found in any tooth.
- They are present as a well defined radiolucency that can vary in size from few mm to several cm.
- If infected secondarily then patients will become symptomatic.
- These cysts will not expand bone.
- Treatment is surgical curettage.

PARADENTAL CYSTS

(Inflammatory periodontal cyst or collateral cyst)

- Seen on the distal or facial aspect of vital mandibular third molar.
- It is an inflammatory cyst.
- Develops on the lateral surface of tooth root.
- Arises from odontogenic epithelium.
- The cell rests of malassez or reduced enamel epithelium may be the cell of origin.
- This cyst occurrence is rare and should be differentiated from lateral periodontal cyst.

Clinical Features

- Well defined radiolucency with non-widening of periodontal space and the lesion was superimposed on buccal root surface.

Histologic Features

- Shows hyperplastic non-keratinized stratified squamous epithelium.
- Intense inflammatory cell infiltrate seen in the fibrous capsule.

Treatment

- Enucleation and extraction of associated molar.
- Contains intensely inflamed connective tissue and epithelial lining.

ODONTOGENIC TUMORS

- Tumors that arise from odontogenic residues, i.e. odontogenic epithelium and/or ectomesenchyme with variable amount of dental hard tissues formed in same sequence as normal tooth development.

Classification

- WHO (1992) with subdivisions in each category (White DK 2004).

According to type of Odontogenic Tissues Involves

A. Benign

- I. *Odontogenic epithelium without odontogenic ectomesenchyme.*
 - Ameloblastoma.
 - Squamous odontogenic tumor.
 - Adenomatoid odontogenic tumor.
 - Calcifying epithelial odontogenic tumor.
- II. *Odontogenic epithelium with odontogenic ectomesenchyme with (or) without hard tissue formation.*
 - Ameloblastic fibroma.
 - Ameloblastic fibrodentinoma.
 - Ameloblastic fibrodontoma.
 - Odontoameloblastoma.
 - Complex odontoma.
 - Compound odontoma.
 - Calcifying odontogenic cyst.
- III. *Odontogenic ectomesenchyme with (or) without included odontogenic epithelium.*
 - Odontogenic fibroma.
 - Myxomas.
 - Cementoblastoma.

B. Malignant

- I. *Odontogenic carcinomas.*
 - Malignant ameloblastoma.
 - Primary intraosseous carcinoma.
 - Clear cell odontogenic carcinoma.
 - Ghost cell odontogenic carcinoma.
- II. *Odontogenic sarcomas.*
 - Ameloblastic fibrosarcoma.
 - Ameloblastic fibrodedentinosarcoma.

AMELOBLASTOMA

(Admantinoma)

Defined by Robinson as

- Usually unicentric, nonfunctional, intermittent in growth, anatomically benign and clinically persistent.
- Second most common odontogenic neoplasm.

Pathogenesis

Derived From

- Cell rests of enamel organ
 - Remnants of Dental Lamina.
 - Epithelial Rest of Malassez.
- Epithelium of odontogenic cysts (esp. Dentigerous cyst).
- Disturbances of developing enamel organ.
- Basal cells of surface epithelium of jaws.
- Heterotopic epithelium in other parts of the body (esp. pituitary gland).

Clinical Features

- Age → 10 to 90 years (Average → 33 to 39 years).
- No significant sex predilection.
- Site → 80% cases mandible premolar, anterior regions.
- Molar–angle – Ramus.
(3 times more common)
- Peripheral ameloblastoma (Extrasosseous).
- Resembles intraosseous type, but which occurs in the soft tissue outside alveolar bone.
- *Origin*: Surface epithelium.
- Remnants of dental lamina.
- *Age*: 23 to 82 years.
- *Sex*: Male predilection.
- *Site*: 2:1 (Mandible to Maxilla).

Differentiation from Intraosseous Type

- Occurs as nodules on the gingiva.
- Innocuous.
- Lacks persistent invasiveness.
- Lesser tendency to recurrence.

Treatment

- Surgical excision.

PITUITARY AMELOBLASTOMA

(Craniopharyngioma, Rathke's Pouch Tumor)

- Neoplasm involving central nervous system.
- Pseudoencapsulated mass destroying the pituitary gland.
- Age : 13-23 years.
- Origin : Unobliterated portions of fetal craniopharyngeal duct, which is derived from Rathke's pouch.

Histology

- Irregular calcified masses.
- Foci of metaplastic bone (or) cartilage.
- Islands and Nests of "Ghost cells".
- Cystic formation.

Adamantinoma of Long Bones

- Bears a superficial resemblance to ameloblastoma of jaws.
- Site: 90% tibia.
- Origin is not related to that of the jaws.

Roentgenographic Features

- Multilocular radiolucency of jaw.
- Compartmented appearance of tumor. (Due to septa of bone extending into radiolucent tumor.)
- Some cases → unilocular lesion.
- Periphery of the lesion → Smooth, but may not be borne out at the time of the surgery.
- Thinning of cortical plate.
- Term used "cystic ameloblastoma".

Histology

(Six Subtypes)

- Follicular ameloblastoma
- Acanthomatous ameloblastoma
- Granular cell ameloblastoma
- Basal cell ameloblastoma
- Desmoplastic ameloblastoma
- Plexiform ameloblastoma.

Follicular (Simple) Type

- Small, discrete islands of tumor composed of peripheral cuboidal (or) columnar cells, with, central.
- Cells resembling “stellate reticulum”.

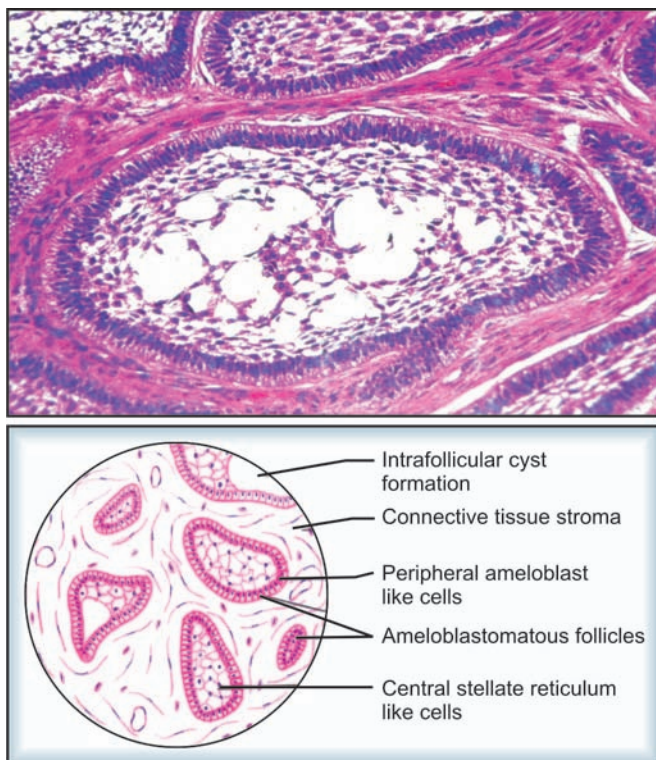


Fig. 14.4: Follicular ameloblastoma

- Cyst formation is common due to breakdown of stellate reticulum like tissue.

Plexiform Ameloblastoma

- Ameloblasts like tumor cells arranged in irregular masses, as a network of interconnecting strands of cells.
- Each of these strands is bounded by layer of columnar cells and between these layers -> stellate reticulum like cells (which is less prominent).
- Areas of cystic degeneration – common.

Acanthomatous Type

- Cells occupying position of stellate reticulum undergo squamous metaplasia, sometimes with keratin formation.
- Usually occurs in follicular type.

Granular Cell Type

- Marked transformation of cytoplasm, into a coarse, granular, eosinophilic appearance, which later involves peripheral columnar (or) cuboidal cells.
- Aggressive lesion with marked tendency to recurrence.

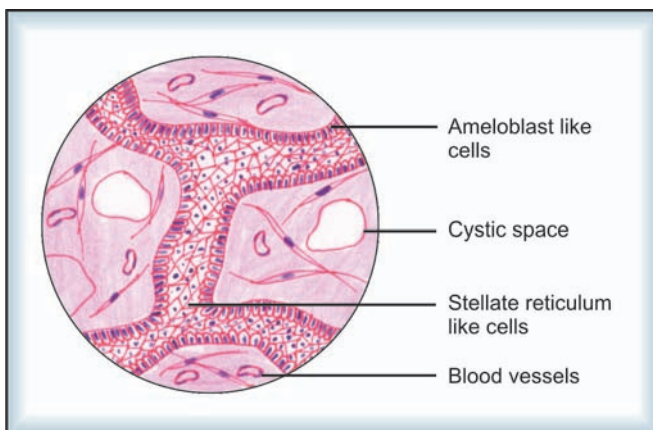


Fig. 14.5: Plexiform ameloblastoma

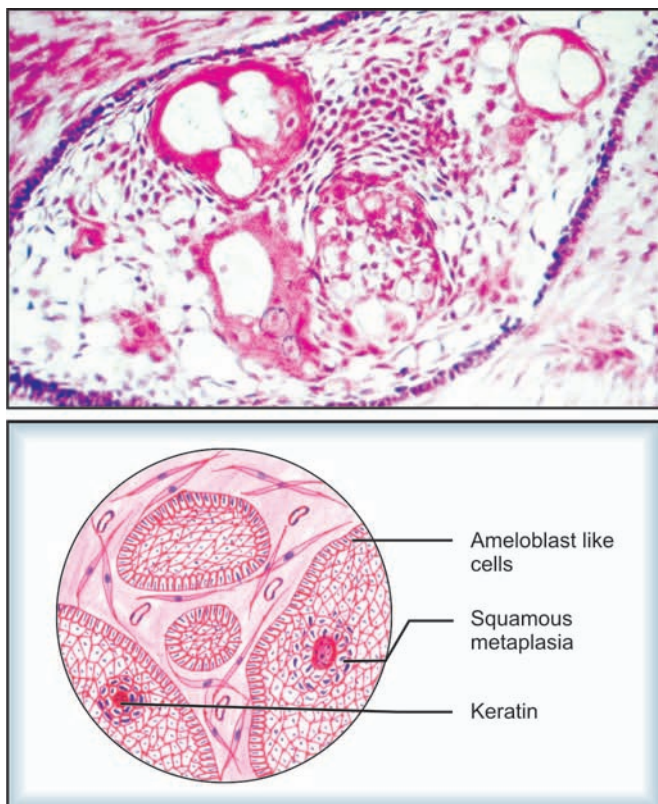


Fig. 14.6: Acanthomatous ameloblastoma

Basal Cell Type

- Resembles basal cell carcinoma of skin.
- Tumor cells are more primitive and less columnar, arranged in sheets.

Desmoplastic Type

- Thin strands (or) cords of epithelium in a dense, collagen stroma that appears hyalinized and hypocellular.
- Periphery of strands are flattened (or) cuboidal cells.

UNICYSTIC AMELOBLASTOMA

- Second and less for frequent growth pattern seen in intraosseous type (6%).
- Age → 22 years.
- Associated with an impacted tooth.

Characteristic Features

- Lining epithelium exhibits **Vickers and Gorlin** criteria, representing early ameloblastomatous changes in the dentigerous cyst.
- Nodules of tumor projecting intraluminally
- Lining proliferating into connective tissue wall.
- Islands of ameloblastoma occurring isolated in connective tissue wall.

Vickers and Gorlin Criteria Includes

- Epithelium shows basal cell layer, composed of columnar cells.
- Displaying hyperchromatic, palisaded nuclei, reversal of polarity, subnuclear vacuole.
- Many instances this resembles Plexiform type, hence termed as “Plexiform Unicystic Type”.

Treatment and Prognosis

- Radical and conservative surgical excision.
- Curettage.
- Electrocautery.
- Radiation therapy.

CALCIFYING EPITHELIAL ODONTOGENIC TUMOR

(Pindborg tumor)

Origin

- Reminiscent of cells in stratum intermedium layer of enamel organ.
- Remnants of primitive dental lamina.

Clinical Features

- Age: 40 years
- Sex: Slight female predilection.
- Site: 2:1 -> Mandible to Maxilla.
- Asymptomatic, swelling, (52% associated with an unerupted (or) impacted tooth).

Radiological Features

- Diffuse (or) well – circumscribed unilocular radiolucent area.
- Some cases – show radiolucency with radiopacity.
(due to irregular bony trabeculae traversing radiolucent areas)
- Producing honeycomb appearance.
- Scattered flecks of calcifications, throughout radiolucency giving a “snow driven appearance”.
- Thinning of buccal and lingual cortical plates.

Histological Features

- Composed of polyhedral, epithelial cells, packed in sheets.
- Cells are arranged in cords (or) rows, mimicking adenocarcinoma.
- Tumor cells have a outlined cell border with a finely granular, eosinophilic cytoplasm and intercellular bridges are often prominent.
- Nuclei are pleomorphic, with giant nuclei and multinucleation.
- A well-recognized form is a “clear – cell variant”.
- Tumor cells exhibit a “clear vacuolated cytoplasm”.
- Nucleus remains round (or) oval in center of cells (or) be flattened against cell membrane.
- Presence of a homogeneous, eosinophilic substance interpreted as amyloid, comparable glycoprotein, basal lamina, keratin, enamel matrix.
- Stains metachromatically with crystal violet, positive with congo red, similar to amyloid.
- Presence of calcification in large amounts in form of Liesegang rings.

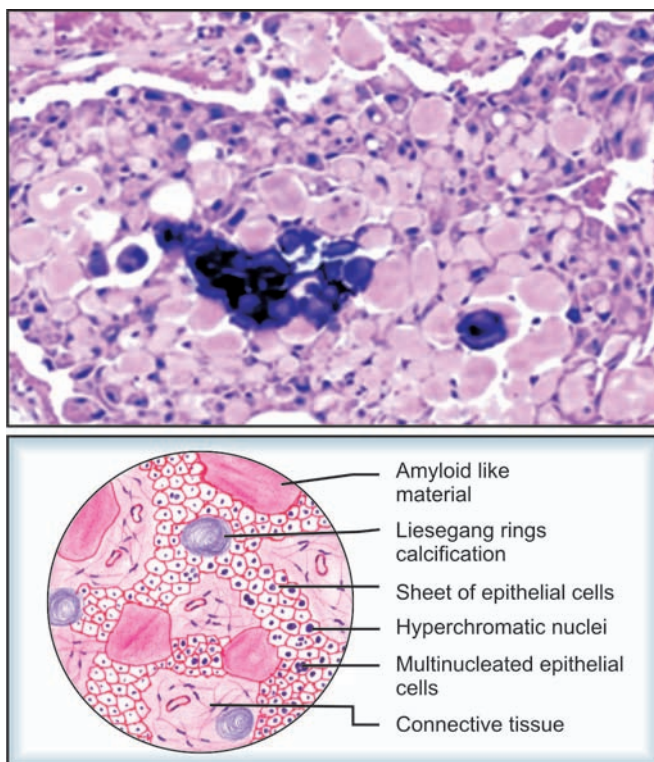


Fig. 14.7: Calcifying epithelial odontogenic tumor

Treatment

- Enucleation (or) curettage followed by judicious removal of bone adjacent to tumor. (thin layer).
- Enlarged tumors → hemimandibulectomy (or) hemimaxillectomy.
- External beam radiation therapy and adjunctive chemotherapy.

ADENOMATOID ODONTOGENIC TUMOR

- Benign neoplasm commonly associated with an unerupted Maxillary cuspid.

Histogenesis

- Etiology – unknown.
- Associated most often with an unerupted (or) impacted tooth.
- Origin – odontogenic.

Clinical Features

- Age : 18 years (5-53 years).
- Sex: Female predilection (64%).
- Site: Maxilla – 65% (Anterior part of Jaws - 76%).
- Mandible: 35% (Anterior to cuspids).
- 74% cases associated with unerupted (or) impacted tooth.
- Size: 1.5 to 3 cm.
- Asymptomatic swelling, usually within jaws (or) gingiva.
- Peripheral lesion: Painless, gingival – colored mass.
- 10 times more prevalent in maxilla.
- Female: male ratio-14:1.

Radiological Features

- Presents as a well-demarcated. Unilocular radiolucency, with smooth, corticated border.
- Most lesions are pericoronal, but radiolucency may extend beyond cemento-enamel junction.
- Intralesional radiopacity may be identified.
- Divergence of roots, displacement, root resorption.

Histologic Features

Macroscopically

Soft, rough spherical mass, with fibrous capsule.

Microscopically

- Multinodular proliferation of spindle, cuboidal, columnar cells, in forms as.
 - Duct like structures
 - Eosinophilic material.

Calcifications

- *Distinctive feature:* Duct like structures with lumina of varying sizes lined by a single layer of cuboidal (or) columnar epithelial cells, with polarization of nuclei away from lumen.

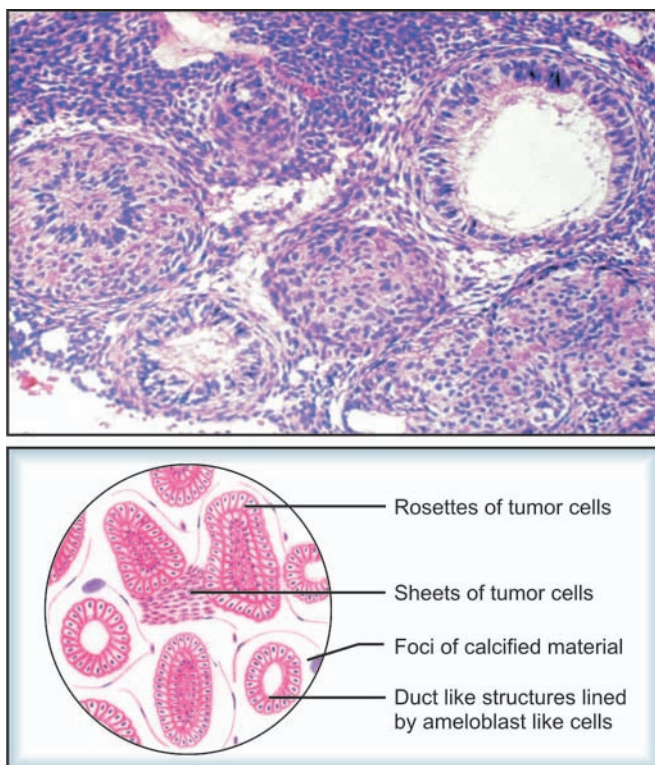


Fig. 14.8: Adenomatoid odontogenic tumor

- These ducts are lined by eosinophilic rims (Hyaline rings).
- Small amounts of eosinophilic material (or) calcifications are present between stellate reticulum like cells (or) polygonal cells.
- Cystic component is seen in certain tumors.
- *Some AOT:* Contain dysplastic dentin.
- Dentinoid.
- Osteodentin.
- Irregular calcified bodies with concentric pattern (Liesegang rings).
- Supporting stroma is loose, hypocellular, fibrovascular.

Treatment

- Conservative surgical excision.

SQUAMOUS ODONTOGENIC TUMOR

- Mistaken histologic identification as Acanthomatous ameloblastoma (or) well-differentiated squamous cell carcinoma.

Histogenesis

- Rests of malassez – for lesions adjacent to lateral root surface.
- Dental lamina → lesions associated with crown.

Clinical Features

- *Age:* 19 to 31 years.
- *Sex:* Slight male predilection.
- *Site:* Maxilla – (Incisor-cuspid) – area.
- Mandible – (Bicuspid-molar) area.
- Symptoms – Mobility of involved teeth and pain in those teeth.
- Tenderness to percussion.
- Abnormal sensations.

Radiological Features

- Semicircular (or) roughly triangular radiolucent area, with (or) without sclerotic border.

Histologic Features

- Islands of mature squamous epithelium, without a peripheral palisaded (or) polarized columnar layer.
- Peripheral layer is quite flattened.
- Squamous cells are uniform, with occasional individual cell keratinization.
- Macrocyst formation.
- Epithelial islands with laminar calcifications, globular, hyaline, eosinophilic structures.

Differential Diagnosis

- Acanthomatous variants of Ameloblastoma.
- Desmoplastic variants of Ameloblastoma.
- Squamous cell Carcinoma.
- Dentigerous cyst, radicular cyst exhibit foci of squamous odontogenic tumor proliferation.

Treatment

- Enucleation
- Local excision
- Curettage

**ODONTOGENIC EPITHELIUM WITH ODONTOGENIC
ECTOMESENCHYME WITH (OR) WITHOUT
HARD TISSUE FORMATION**

AMELOBLASTIC FIBROMA

- Characterized by simultaneous proliferation of both epithelial and mesenchymal tissue without formation of enamel and dentin.

Differential Diagnosis

- Immature complex odontoma
- Ameloblastic fibro-odontoma.

Clinical Features

- *Age*: 33 years.
- *Sex*: Slight male predilection.
- *Site*: Molar region of mandible.
- Symptoms → Doesn't infiltrate between trabeculae of bone and
Signs → Gradual expansion of lesion.
- Pain, tenderness (or) mild swelling of jaw.

Radiological Features

- Unilocular (or) multilocular radiolucent lesion with a rather smooth outline with a sclerotic border.
- Most lesions were associated with unerupted teeth.

Histologic Features

- Ectodermal portion consists of scattered islands of epithelial cells in rosettes, finger – like strands, nests and cords.
- Epithelial cells → columnar (or) cuboidal – close resemblance to primitive odontogenic epithelium.

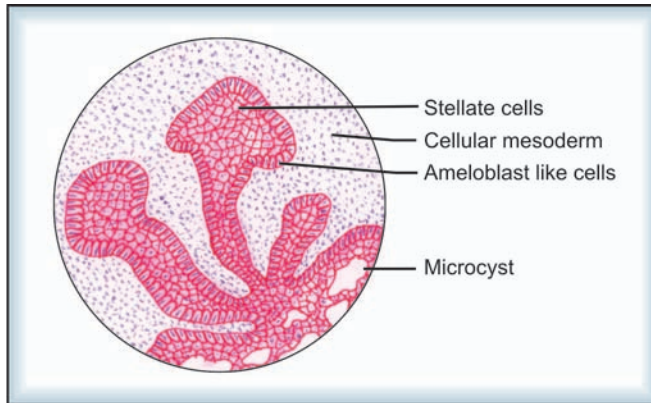


Fig. 14.9: Ameloblastic fibroma

- Mesenchymal component → Primitive connective tissue, showing intertwining fibrils, interspersed with large connective tissues cells resembling Dental papilla.

Treatment and Prognosis

- Conservative surgical excision.
- Little tendency to recurrence.

AMELOBLASTIC FIBRO-ODONTOMA

- Early, undifferentiated complex odontoma and it is a differentiating maturing odontoma.

Clinical Features

- *Sex:* 3.3 :1 Male and female ratio.
- *Age:* 11.5 years. (1-39).
- *Site:* Mandible – 62% (54% in posterior mandible).
- *Maxilla:* 38 percent.
- *Symptoms:* Swelling and failure of tooth eruption.

Radiological Features

- Well, circumscribed lesion, presenting as an expansile radiolucency containing solitary radio-opaque mass representing odontoma.

Histologic Features

- Fingers, cords, strands, and rosettes or primitive odontogenic columnar or cuboidal cells, resembling dental lamina.
- Mesenchymal component → Embryonic connective tissue with delicate fibrils with fibroblasts resembling dental papilla.

Treatment

- Curettage

ODONTOMA

- Growth in which both epithelial and mesenchymal cells exhibit complete differentiation, with the result that functional ameloblasts and odontoblasts form enamel and dentin.
- Enamel and dentin are laid down in an abnormal pattern, because organization of odontogenic epithelium fails to reach a normal state of morphodifferentiation.
- Lesion is composed of more than one type of tissue, hence termed “composite odontoma”.

Types

- Compound composite (Enamel, dentin laid are similar to normal teeth but smaller than them).
- Complex composite (Calcified tissues are an irregular mass with no resemblance even to rudimentary teeth).

Etiology

- Unknown
- Local trauma (or) infection
- Inherited (or) mutant gene (or) interference.

Clinical features

- Sex: Male predilection (59%).
- Age: 14.8 years (Mean age).
- Site: 67% maxilla.
- 33% mandible.
- Compound odontomas → 61% anterior maxilla.
- Complex odontomas 34% anterior maxilla.
- 59% posterior jaws.
- Right side of jaws
- 62% compound type
- 68% complex type.
- Lesion enlarges and produces expansion with consequent facial asymmetry.
- Asymptomatic swelling consisting of unerupted (or) impacted teeth.

Radiological Features

- Situated between roots of teeth and appear as irregular mass of calcified tissue surrounded by a narrow radiolucent band with a smooth outer periphery.
- Structures resembling teeth are seen in some instances.

Histologic Features

- Normal appearing enamel (or) enamel matrix, dentin, pulp tissue, cementum are present.
- Connective tissue capsule around the odontoma is similar in all respects to follicle surrounding a normal tooth.
- Ghost cells are present.

Treatment and Prognosis

- Surgical removal.

AMELOBLASTIC ODONTOMA

- Odontogenic neoplasm characterized by simultaneous occurrence of an ameloblastoma and a composite odontoma.
- These are two separate neoplasms growing in union.

Clinical features

- Occurs at any age. (More common in children).
- *Site*: Common in mandible than Maxilla.
- Slow expanding lesion with facial deformity, (or) asymmetry if left untreated.
- Delayed eruption of teeth.

Radiological Features

- Central bone destruction with expansion of cortical plates.
- Numerous small, radiopaque masses, with resemblance to miniature teeth, in some instances.
- In other instances, irregular calcified masses are seen.

Histologic Features

- Columnar cells, squamous and undifferentiated epithelial cells, as well as ameloblasts, enamel and enamel matrix, dentin. Osteodentin, dentinoid and osteoid material, stellate- reticulum like tissue, dental papilla, bone and cementum and stromal connective tissue are present.
- Basal cell, follicular, plexiform type ameloblastoma is seen.

Treatment and Prognosis

- Conservative curettage (or) enucleation.

ODONTOGENIC ECTOMESENCHYME WITH OR WITHOUT INCLUDED ODONTOGENIC EPITHELIUM

PERIPHERAL ODONTOGENIC FIBROMA

Clinical Features

- *Age*: 5-65 years.
- *Sex*: No predilection.
- *Site*: Mandible predilection.
- Slow growing, firmly attached gingival mass.
- Some contain a calcified stalk (or) radiopaque flecks.

Histologic Features

- Marked cellular fibrous connective tissue parenchyma.
- When numerous, the peripheral odontogenic fibroma has been mistaken for peripheral ameloblastoma.
- Epithelium is deep in the lesion and sometimes “cuffing” calcifications.
- Calcified tissue found may resemble trabeculae of bone (or) osteodentin, cementum like material.
- Highly vascularized, mature connective tissue is seen.

Treatment

- Surgical excision.

CENTRAL ODONTOGENIC FIBROMA

Three Basic Concepts

- Lesion around the crown, small and resembling a dentigerous cyst.
- Lesion of fibrous connective tissue, scattered with odontogenic epithelium islands, with some resemblance to Dental follicle. (Simple type)
- WHO describes it as a fibroblastic neoplasm.
- TYPE with odontogenic epithelium, calcified material.
- Except for location it is identical to peripheral type.

Clinical Features

- *Age:* More frequently in children.
- *Site:* Mandibular predilection.
- Asymptomatic except for swelling of the jaw.

Radiological Features

- Expansile, multilocular radiolucency, similar to ameloblastoma.

Histologic Features

- *Simple type:* Mature collagen fibers, interspersed by plump fibroblasts.
- Inactive odontogenic epithelial islands are present.

- Mature, but quite, cellular fibrous tissue, with few to many islands of odontogenic epithelium.
- Osteoid, dysplastic dentin (or) cementum like material is present.

Treatment and Prognosis

- Surgical excision.

ODONTOGENIC MYXOMA

- Tumor of the jaws, arising from mesenchymal portion of tooth germ, either dental papilla, follicle (or) periodontal ligament.

Clinical Features

- *Age:* 10 to 30 years.
- *Sex:* No predilection.
- *Site:* Predilection for mandible.
- Central lesion of the jaws, which expands bone, destroys cortex.

Radiological Features

- Mottled (or) honeycombed appearance, sometimes expanding radiolucency.
- Displacement of teeth.
- Maxillary tumor → invades antrum.

Histologic Features

- Loosely arranged, spindle-shaped, stellate cells.
- Intercellular substance is mucoid.
- Tumor is interspersed with tiny capillaries, and strands of collagen.
- Presence of hyaluronic acid, and lesser amounts of chondroitin sulfate.
- Presence of “pale, secretory cells” and “dark cell”, containing collagen fibrils.

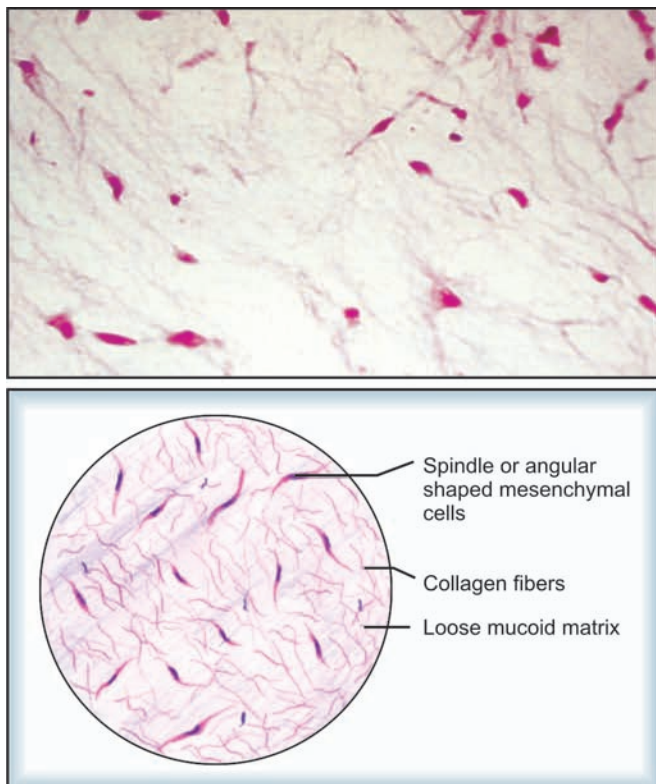


Fig. 14.10: Odontogenic myxoma

Treatment

- Surgical excision followed by cautery.
- Prognosis is good.

MALIGNANT ODONTOGENIC TUMORS

- Odontogenic carcinomas.

METASTASIZING AMELOBLASTOMA

(Malignant ameloblastoma)

- Describes a tumor showing histologic features, of classic ameloblastoma in the gingival (or) jaw, and has metastatic deposits elsewhere.

Clinical Features

- Age: 30 years
- Metastatic nodules seen in Lung (80%)
 - Innocuous granular
 - Cervical lymph nodes (15%).

Histologic Features

- Metastatic ameloblastoma is histologically identical to conventional ameloblastoma.

Treatment

- Surgery.
- Cervical node metastases – Neck dissection.
- Ameloblastic carcinoma.
- Malignant epithelial proliferation, associated with.
- Carcinoma ex-ameloblastoma.
- De Novo Ameloblastic carcinoma (Histologic resemblance).
- Carcinoma ex-ameloblastoma → arises due to “dedifferentiation of Ameloblastoma”
- Follicular (or) Plexiform pattern blends through a narrow transition zone with a hypercellular poorly differentiated carcinoma that demonstrates, sheets of disordered, mitotically – active small basaloid cells with hyperchromatic nuclei, larger polygonal cells, with pale vesicular nuclei.

Denovo Type

- Lacks a component of conventional ameloblastoma.
- Tumor lacks evidence of “Vickers-Gorlin” features.

OTHER TYPES OF CARCINOMAS

- Primary intraosseous carcinoma.
- Solid primary intraosseous carcinoma.
- Cystic primary intraosseous carcinoma.
- Carcinoma ex-dentigerous cyst.
- Carcinoma ex-odontogenic keratocyst.
- Intraosseous mucoepidermoid carcinoma.

Diseases of Bone and Joints

DISEASES OF BONE

- Bone, a dense calcified tissue, is affected by diseases which either involve the entire bony skeleton or only a single bone. Some diseases may have a strict Mendelian pattern of heredity. These diseases of bone may arise at any age.
- “The maxilla and mandible” like other bones, suffer from both the generalized and the localized forms of skeletal diseases. Although the basic reactions are the same, the peculiar anatomic arrangement of teeth embedded partially in bone, through which the bone may be subjected to an unusual variety of stresses, strains, infections, often produces a modified response of bone to the injury.

OSTEOGENESIS IMPERFECTA

(“Brittle Bones”; Fragilitas Ossium; Osteopsathyrosis; Lobstein’s Diseases)

- Osteogenesis imperfecta, a milder condition affecting mesodermal tissues is a serious disease of unknown etiology, is closely related to dentinogenesis imperfecta.
- Even though it represents a hereditary autosomal dominant characteristic generally, autosomal recessive and non-hereditary types also occur.

- The disease in its usual form is present at birth (Congenital or Vrolik's type) although some cases do not arise or are not recognized until later in childhood (tarda or lobsteins type, gravis or levis). The latter variety has also been called osteopsathyrosis.
- Many infants affected with this disorder are stillborn or die shortly after birth.

Clinical Features

- The chief clinical characteristic is the extreme fragility and porosity of the bones which are prone to fracture due to inadequate type-I collagen formation. Even though the fracture heals readily, the new bone is of a similar imperfect quality, Hyperplastic callus formation which may or may not be related to actual fracture, occasionally occurs in some cases. This callus may be so exuberant that it mimics osteosarcoma. True sarcoma arising in such calluses have also been reported.
- Another characteristic clinical feature is the presence of pale, blue sclera. The sclera are abnormally thin, the pigmented choroids shows through and produces the bluish color. Blue sclera may also be seen in Osteopetrosis, Fetal Rickets, Marfan's syndrome, Ehlers - Danlos syndrome as well as in normal infants.
- The other signs and symptoms characteristic of Osteogenesis imperfecta are:
 - Deafness due to osteosclerosis.
 - Abnormalities of the teeth (identical with those of dentinogenesis imperfecta or "hereditary opalescent dentin").
 - Laxity of the ligaments.
 - Anterior and posterior crossbite.
 - A peculiar shape of the skull.
 - An abnormal electrical reaction of the muscles.
 - Open bites
 - Impaction and ectopic teeth.
 - Tendency for capillary bleeding.
 - Class-III malocclusion.

Oral Manifestations

- Since osteogenesis imperfecta is a disturbance of mesodermal tissues, particularly calcified tissues a disturbance in dentin formation only occurs.

- Either both osteogenesis imperfecta and dentinogenesis imperfecta may occur together or dentinogenesis imperfecta may occur without more generalized bone involvement.

Radiographic Features

- Osteopenia, bowing, angulation or deformity of the long bones, multiple fractures and wormian bones (sutural bone) on the skull.

Histologic Features

- The bones have thin cortices, composed of immature spongy bone, while the trabeculae of the cancellous bone are delicate and often show microfractures.
- Osteoblastic activity is imperfect or retarded hence the long bones are deficient in thickness.
- The basic defect appears to lie in the organic matrix with failure of fetal collagen to be transformed into mature collagen but calcification proceeds normally.
- Progressive intermolecular cross - linking of adjacent collagen molecules is also defective in this disease.
- Defective microvascular system and decreased collagen fibrils diameter have also been observed.

Treatment

- No known treatment. Clinical improvement following onset of puberty in girls has been noted.

INFANTILE CORTICAL HYPEROSTOSIS

(Caffey's Disease; Caffey - Silverman Syndrome)

Etiology

- An embryonal osteodysgenesis consequent to a local defect in the blood supply to the area.
- An inherited defect of arterioles supplying the affected part results in hypoxia producing focal necrosis of overlying soft tissues and periosteal proliferation.
- An allergic phenomenon in which edema and inflammation produce periosteal elevation and subsequent deposition of calcium.

- A hereditary pattern, transmitted as an autosomal dominant, trait probably with incomplete penetrance.

Clinical Features

- Characterized by the development of tender, deeply placed soft
 - Tissue swellings and cortical thickening or hyperostosis involving skeletal bones.
- This disease usually arises during the first 3 months of life, but it may not appear before the second year.
- Commonly affected are the mandible, clavicles. Mandibular involvement, which is manifested as a facial swelling is a striking feature.
- lower extremity involvement is more frequent in the familial form.
- The calvarium, scapula, ribs and tubular bones of the extremities, including metatarsals also show hyperostosis.
- Other signs and symptoms include fever, hyperirritability, pseudo paralysis, dysphagia, pleurisy, anemia, leukocytosis, monocytosis, elevated sedimentation rate, increased serum alkaline phosphatase.

Oral Manifestations

- Residual asymmetric deformity of the mandible usually in the angle and ramus area.
- Severe malocclusion but rarely.

Radiographic Findings

- Involvement may be unilateral or bilateral and is manifested as a gross thickening and sclerosis of the cortex due to an actively proliferating periosteum.
- Since the hyperostosis usually later follows the clinical appearance of the soft-tissue swelling, there is inability to demonstrate roentgenographic evidence of the diseases in the early stage.

Treatment and Prognosis

- Active manifestation regress without treatment.

CLEIDOCRANIAL DYSPLASIA

(Marie and Sainton's Disease; Scheuthauer- Marie - Sainton Syndrome; Mutational Dysostosis)

- A disease of unknown etiology which is often but not always hereditary.
- In inherited cases, it appears as a dominant Mendelian characteristic, transmitted by either sex.
- In sporadic cases, it appears as a recessively inherited disease.

Clinical Features

- Characterized by abnormalities of the skull, teeth, jaws and shoulder girdle occasional stunting of the long bones.
- Fontanels often remain open or exhibit delayed closing.
- Sutures may remain open wormian bones are common.
- Sagittal suture is characteristically sunken giving the skull a flat appearance. Frontal, parietal, occipital bones are prominent and the paranasal sinuses are underdeveloped and narrow.
- The head is brachycephalic, or wide and short, the transverse diameter of the skull being increased.
- The defect of the shoulder girdle ranges from complete absence of clavicles or even a simple thinning of one or both clavicles or partial absence. Because of this clavicle disturbance the patients have an unusual mobility of the shoulders may be able to bring their shoulders forward until they meet in the midline.

Radiographic Findings

- Characterized by widely patent anterior fontanel and sutures with wormian bones in cranion.
- Clavicles are reduced to single or double fragments.

Oral Manifestation

- High narrow arched palate.
- Actual cleft palate.
- Under developed maxilla.
- (Lacrimal zygomatic bones are also underdeveloped).

- Prolonged retention deciduous teeth subsequent delay in eruption of succedaneous teeth.
- Roots of the teeth are short, thinner and usually deformed.
- Absence or paucity of cellular cementum on the roots of permanent teeth which may be related to the failure of eruption. But there is no increased thickening of a cellular cementum.
- Numerous unerupted supernumerary teeth (X-ray finding).
- Rarely/ partial anodontia.

Treating and Prognosis

- No specific treatment.
- Recently, a multidisciplinary approach utilizing the pedodontist, orthodontist and oral surgeon is done.

CRANIOFACIAL DYSOSTOSIS

(Crouzon Disease or Syndrome)

- Craniofacial dysostosis is the commonest form craniosynostotic syndrome occurring without syndactyly. If it occurs with syndactyly, it is called Apert syndrome.

Clinical Features

- All the clinical signs are all due to the early synostosis of the sutures.
- Protuberant frontal region with an anteroposterior ridge overhanging the frontal eminence and often passing to the root of the nose - Triangular frontal defect.
- Maxillary hypoplasia with mandibular prognathism.
- High arched palate, frog face.
- Palatal cleft (rare).
- Exaggerated facial angle.
- Parrot's beak type of nose.
- Hypertelorism, exophthalmoses with divergent strabismus, optic neuritis and choked disks resulting in blindness.
- Spina bifida occulta.
- Mentality may or may not be retarded.
- Malocclusion, headache and convulsion.

Treatment and Prognosis

- Craniectomy at very early stage to provide spaces for rapidly developing brain.
- Surgical procedures were basically designed to improve the cosmetic appearance and vision of patient.

MANDIBULOFACIAL DYSOSTOSIS

(Treacher Collins Syndrome-Franceschetti Syndrome)

- The mandibulofacial dysostosis includes a group of closely related defects of the head and face, often hereditary or familial, following an irregular form of dominant transmission.

Clinical Features

- Antimongoloid palpebral fissures with a coloboma of the outer portion of the lower lids and deficiency of the eye lashes.
- Hypoplasia of the facial bones, especially of the malar bones and mandible.
- Malformation of the external ear and occasionally of the middle and internal ears.
- Macrostomia, high palate and abnormal position and malocclusion.
- Blind fistulas between the angles of the ears the angles of the mouth.
- A typical hair growth in the form of a tongue - shaped process of the hairline extending towards the cheeks.
- Facial clefts and skeletal deformities.
- Patient are described as being “bird like or fish like” in nature.
- The syndrome is a result of retardation or failure of differentiation of maxillary mesoderm at or after the 50 mm stage of the embryo.

Radiographic Findings

- Agenesis of the malar bones with non-fusion of the Zygomatic arches as well as absence of the palatine bones.
- Hypogenesis/agenesis of mandible.
- Underdeveloped paranasal sinuses.
- Infantile sclerotic mastoids.

- Absence of auditory ossicles.
- Deficient cochlear and vestibular apparatus.

Treatment and Prognosis

- No treatment, but prognosis is good.

PIERRE-ROBIN SYNDROME

(Robin anomalad)

- Pierre-Robin syndrome is a non-specific anomaly which may occur either as an isolated defect or as a part of a broader group of malformation.
- The isolated defect is a sporadic or non-genetic condition with a very low recurrence risk.
- Pierre-Robin syndrome is associated with other genetic syndromes and has a very high recurrence risk.
- An anomalad is a malformation together with its subsequently derived structural changes, the 1° defect setting off a series of 2° or even 3° events resulting in multiple anomalies.
- The Pierre-Robin syndrome consists of
 - Cleft palate
 - Micrognathia
 - Glossoptosis.
- In this syndrome the primary defect lies in arrested development and ensuing hypoplasia of the mandible, producing the characteristic “bird facies” which in turn prevents the normal descent of the tongue between the palatal shelves, resulting in cleft palate. Hence cleft lip does not occur along with cleft palate.
- Respiratory difficulty occurs as a result of jaw malformation which is due to the failure of support of tongue musculature because of micrognathia, allowing the tongue to fall down backward, partially obstructing the epiglottis.
- Congenital heart defects, skeletal defects, ocular lesions and mental retardation may also be present.

MARFAN'S SYNDROME

(Marfan's-Achard syndrome; arachnodactyly)

- Marfan's syndrome, a hereditary autosomal dominant condition, is a disease of connective tissue related to a defective organization of collagen which is abnormally soluble.

Clinical Features

- Dolichostenomelia or disproportionately long thin extremities.
- Arachnodactyly or spidery fingers.
- Long narrow skull and face.
- Hyperextensibility of joints with habitual dislocations.
- Kyphosis or scoliosis and flatfeet.
- Bilateral ectopia lentis (due to weakening or rupture of the suspensory ligaments).
- Myopia.
- Cardiovascular complication - Aortic aneurysm, aortic regurgitation, valvular defects, cardiomegaly.

Oral Manifestations

- High arched palatal vault.
- Bifid uvula.
- Malocclusion.
- Multiple odontogenic cysts.
- Temporomandibular dysarthrosis.

DOWN SYNDROME

(Trisomy 21 syndrome, Mongolism)

- Down syndrome is the most common chromosomal abnormality with associated subnormal mentality.
- There are three types of Down syndrome.
 - Typical trisomy 21
 - Translocation type
 - Chromosomal mosaicism.

Clinical Features

- Flat face, mid face hypoplasia.
- Large anterior fontanel.
- Open sutures.
- Small slanting eyes with epicanthal folds.
- Open mouth.
- Prognathism.

- Sexual underdevelopment.
- Hypermobility of joints.
- Poorly developed paranasal sinuses and malformed upper respiratory tract.
- Cardiac abnormalities.
- Increased incidence of acute leukemia in children.

Oral Manifestation

- Macroglossia with protruded tongue, as well as fissured tongue or pebbly tongue (enlarged papillae).
- Enamel hypoplasia.
- High arched palate.
- Microdontia.
- Malocclusion.
- Everted, thick dry crusted lip.
- Delayed tooth eruption.
- Gross plaque accumulation.
- Low caries activity (high bicarbonate content and pH of saliva).
- Rapidly progressive periodontal disease.

Treatment

- No specific treatment.

OSTEOPETROSIS

(Marble Bone Disease; Albers - Schonberg Disease; Osteosclerosis Fragilis Generalisata)

- Osteopetrosis is a rare genetic disease in which the bones become solidified and dense but brittle.
- There are two main types.
 - A clinically benign dominantly inherited form.
 - A clinically malignant recessively inherited form.

Clinical Features

Benign dominant osteopetrosis

- Develops later in life.
- Mostly asymptomatic.
- Multiple pathologic fractures.

- Bone pain.
- Cranial nerve palsy (due to narrowing of the cranial foramina by bone deposition which results in nerve impingement).
- Osteomyelitis.

Malignant recessive osteopetrosis

- More severe may be of congenital or neonatal in origin.
- Optic atrophy.
- Hepatosplenomegaly.
- Poor growth.
- Frontal bossing.
- Pathologic fractures.
- Loss of hearing.
- Facial palsy.
- Genu valgum.
- Death, as a result of anemia or secondary infection.

Oral Manifestations

- Reduced medullary spaces of the jaws with an increased chance for osteomyelitis if infection enters bone.
- Jaw fracture during extraction due to fragility of bone.
- Enamel hypoplasia.
- Microscopic dentinal defects.
- Arrested root development.
- More prone to dental caries.
- Retardation of tooth eruption due to bone sclerosis.

Radiographic Findings

- A diffuse, homogenous, symmetrically sclerotic appearance of all bones with clubbing and transverse striations of the ends of the long bones.
- Medullary cavities are replaced by bone and the cortex is thickened.

Lab Findings

- Myelophthitic anemia due to the displacement of hemopoietic marrow tissue by bone.
- Increased serum acid phosphate (in benign dominant type).

Histologic Features

- Endosteal production of bone with an apparent lack of physiologic bone resorption (osteoblasts are prominent, osteoclasts are rare).
- In polarized light, there is deficient collagen matrix fibrils which seldom crossed from one osteone to another. This deficient fibrils is the cause for increased tendency to fracture.

Treatment

- No effective treatment.
- Bone marrow transplantation may be of some use.

ACHONDROPLASIA

(Chondrodystrophia Fetalis)

- A chondroplasia is a condition resulting due to a disturbance in endochondral bone formation and is transmitted as an autosomal dominant character which results in a characteristic form of dwarfism.
- Disease begins in utero and may be diagnosed before parturition and has a high mortality rate.

Clinical Features

The following are the characteristic features of chondroplastic dwarf:

- Short.
- Short and thickened muscular extremities.
- Bowed legs.
- Brachycephalic skull.
- Small hands with stubby fingers.
- Lumbar Lordosis with prominent buttocks.
- Protruding abdomen.
- Limitation of joint movement.
- Elbows can not be straightened.
- Normal intelligence.
- Unusual strength and agility.
- Disproportionate size of the head in relation to the remainder of the body.

Oral Manifestations

- Retruded maxilla, due to restriction of growth of the base of skull.
- Malocclusion, because of disproportionate jaw sizes.
- Congenital missing teeth with disturbance in shape.

Radiographic Findings

- Thickening or mild clubbing of the ends of long bones.
- Premature fusion of the bones at the base of the skull leading to a narrow foramen magnum.

Histologic Features

- Disturbances in the epiphyseal cartilage of long bones, ribs and certain membrane bones of the base of skull.
- Retardation or aplasia of the zone of provisional calcification of endochondral growth.

Treatment and Prognosis

- No treatment. If the patient survives his first few years, then there is a chance for normal life.

PAGET'S DISEASE OF BONE

(Osteitis deformans)

Etiology

- The etiology of Paget's disease is not known even though various theories have been proposed.
- An inflammatory origin.
- A circulatory disturbance - Bone is extremely vascular and the vessels are similar to arteriovenous aneurysms.
- A breakdown in the normal mechanism of creeping replacement to which bone is subjected.
- An infection by a "slow" virus (one which produces a disease with a prolonged incubation period prior to manifestation of the illness).

Clinical Features

- Person past middle age are commonly affected.
- Bone pain.
- Severe headache.
- Deafness (due to involvement of the petrous part of temporal bone with compression of cochlear N. in its foramen).
- Blindness (due to involvement of optic N.).
- Facial paralysis (due to compression of Facial N.).
- Dizziness, weakness.
- Mental disturbance.
- Progressive skull enlargement.
- Deformities of spine, femur, tibia - Bowing of legs, broadening and flattening Simian appearance of chest and spinal curvature.

Waddling Gait

- Grotesque facial pattern.
- Bones are warm to touch due to increased vascularity.
- Pathologic fracture.

Oral Manifestations

- Progressively enlarging maxilla.
- Widened alveolar ridge.
- Flattened palate.
- Teeth become loose and migrate and show spacing.
- As the disease progresses, the mouth remains open, since the lip are too small to cover the enlarged jaws.
- Inability to wear dentures due to jaw enlargement.

Radiographic Findings

- Characterized by an initial phase of deossification and softening followed by a bizarre, dysplastic type of re-ossification.
- Large isolated lesion in the skull are referred to as "Osteoporosis circum-scripta".
- Osteoblastic areas which are patchy in distribution but with a variation in radiodensity, present the characteristic "Cotton - wool" appearance.
- Loss of trabeculation.
- Hypercementosis.

- Loss of a well defined lamina dura around the teeth.
- Very rarely, root resorption may occur.

Lab Findings

- Elevated serum alkaline phosphatase, to extreme limits.

Histologic Features

- “Mosaic” bone formation which indicates the appearance of bone which has been partially resorbed then repaired, producing deeply staining hematoxyphilic reversal line which indicates alternate resorptive and formative phases leading to a “jig saw puzzle” appearance.
- Fibrous marrow with inflammatory edema.
- Focal collections of lymphocytes.

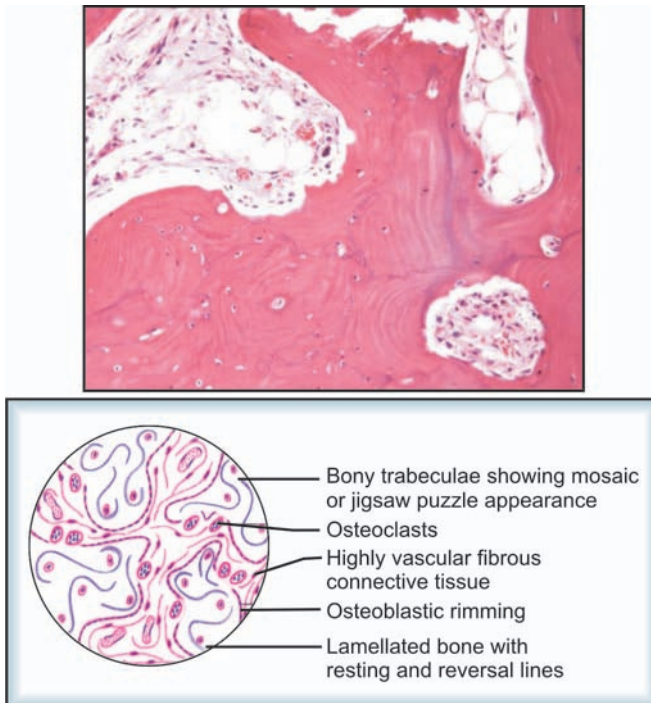


Fig. 15.1: Paget's disease of bone (osteitis deformans)

Treatment and Prognosis

- No specific treatment.
- Vitamin, hormone and radiation therapy are utilized (calcitonin, diphosphonates, cytotoxic antibiotics are used).
- Most serious complication of Paget's disease is development of osteosarcoma.

FIBROUS DYSPLASIA OF BONE

- Fibrous dysplasia of bone is a fibro osseous lesion of unknown etiology, uncertain pathogenesis and diverse histopathology.
- There are two forms of fibrous dysplasia depending on whether one or more bone is involved.
 - Monostotic.
 - Polyostotic.

(a) Monostotic Fibrous Dysplasia of the Jaws

- Does not manifest extra skeletal lesion.
- Jaws are more frequently affected.

Etiology

- Etiology is unknown although various factors have.
- Aberrant activity in the bone-forming mesenchymal tissue.
- Local infections or trauma.
- A peculiar reparative reaction on the part of bone to any one of a variety of injuries.
- An autosomal recessive disorder (congenital).

Clinical Features

- More common in children and young adults with a slight female predilection.
- Painless swelling or bulging of the jaw is the first clinical sign (Maxilla - Labial/Buccal Plate, Mandible - inferior border).
- Malalignment, tipping/displacement of teeth.
- Tenderness.
- Intact mucosa over the lesion.

- Fibrous dysplasia of the maxilla is the most serious form and common in children. It has the following characteristic features:
 - Not well circumscribed.
 - Extends locally to involve the maxillary sinus, the zygomatic process, the floor of the orbit, extends back towards the base of the skull.
 - Malocclusion bulging of the canine fossa or extreme prominence of the Zygomatic process leading to facial deformity.
 - Also referred to as “craniofacial fibrous dysplasia” since it involves craniofacial complex.

Radiographic Findings

- Extremely variable radiographic appearance and presents three basic patterns.
 - Small unilocular or larger multilocular radiolucency with a well - circumscribed border containing a network of fine bony trabeculae.
 - Increased trabeculation with more opacity and typically mottled in appearance.
 - Opaque with many delicate trabeculae giving a “ground - glass” or “peaud’ orange” appearance. Not well circumscribed but blends into the adjacent normal bone.
- In craniofacial fibrous dysplasia, there is characteristic roentgenographic thickening of the base of the skull.

Histologic Features

- Histologically, the lesion is fibrous with proliferating fibroblast in a compact stroma of interlacing collagen fibers.
- Irregular trabeculae which are coarse woven bone are scattered throughout with no definite pattern. Some may be C-shaped or chinese character - shaped.
- On maturation the lesional tissue may show lamellae bone.

Treatment and Prognosis

- Surgical treatment either by block - resection (since not circumscribed) or conservative removal.
- Very rarely, malignant transformation especially osteosarcoma have been reported.

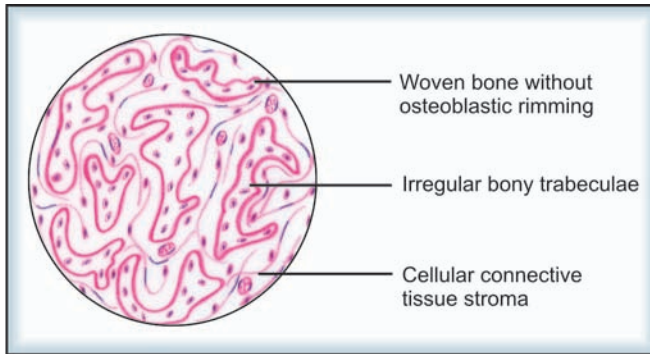


Fig. 15.2: Fibrous dysplasia

- Also sarcomas have been reported following X-ray radiation therapy.

(b) Polyostotic Fibrous Dysplasia

- Two types of Polyostotic fibrous dysplasia are described Jaffe's type - involving a variable number of bones, associated with pigmented lesions of the skin or "Cafe-au-lait" spots. Albright's syndrome—more severe lesion involving almost all skeletal bones associated with pigmented lesions of skin and also endocrine disturbances of varying types.

Clinical Features

- Early manifestation.
- Bowing/thickening of long bones (often unilateral).
- Aching recurrent bone pain.
- Facial skull bones are involved frequently leading to asymmetry.
- Clavicles, pelvic bones, scapulae, long bones, metacarpals and metatarsals are involved.
- Spontaneous fracture.
- Precocious puberty in females.
- Vaginal bleeding.
- Pituitary, thyroid, parathyroid and ovarian glands are involved.
- Multiple, intramuscular, soft tissue myxomas may also occur as an extraskeletal manifestation.

Oral Manifestation

- Expansion and deformity of the jaws.
- Eruption pattern of the teeth is disturbed.

Radiographic Findings

- Medullary portions of the bone are rarefied present irregular trabeculations, often a multilocular cystic appearance.
- Thinned and expanded cortical bone.

Lab Findings

- Increased serum alkaline phosphatase level.
- Premature secretion of pituitary follicle stimulating hormone.

Histologic Features

- Fibrillar connective tissue with numerous trabeculae of coarse, woven fiber bone, irregular in shape but evenly spaced, showing no relation to functional patterns.

Treatment and Prognosis

- Mild cases can be surgically treated.
- Severe cases can be treated with X-ray radiation but may lead to radiation induced sarcomas.
- Malignant transformation may occur (Osteosarcoma).

CHERUBISM

(Familial fibrous dysplasia of the jaws; Disseminated juvenile fibrous dysplasia; Familial multilocular cystic disease of the jaws; Familial fibrous swelling of the jaws; Hereditary fibrous dysplasia of the jaws)

- Cherubism is an uncommon disease inherited as an autosomal dominant trait.

Clinical Features

- Manifests in early childhood.
- A progressive, painless, symmetric swelling of the jaws, producing the typical chubby face or cherub.

- Jaws are firm and hard.
- Regional lymphadenopathy.
- Enlarged palate.
- Premature, spontaneous shedding of deciduous dentition.
- Defective permanent dentition (either absent or displacement or lack of eruption).

Radiographic Findings

- Extensive bilateral destruction of bone with expansion and severe thinning of the cortical plate.
- Multilocular appearance of the body of the bone.
- Cortical perforation may occur.
- Numerous unerupted displaced teeth which may appear to be floating in cyst like spaces.

Histologic Features

- Presence of large multinucleated giant cells in a loose, delicate fibrillar connective tissue stroma with many fibroblasts and small blood vessels.
- Sprinkling of inflammatory cells may be seen.
- Epithelial remnants from the developing teeth.
- A peculiar, perivascular, eosinophilic cuffing of the small capillaries.

Treatment and Prognosis

- Lesions regress with skeletal maturation and normal facial contour may be restored.
- Radiation therapy is definitely contraindicated.

GENERALIZED CORTICAL HYPEROSTOSIS

(Van Buchem's disease; Endosteal hyperostosis)

- A hereditary autosomal recessive condition with excessive deposition of endosteal bone throughout the skeleton.

Clinical Features

- Does not manifest till adulthood.

- Face appear swollen with widening of the angles of mandible and at the bridge of nose.
 - Loss of visual acuity
 - Loss of facial sensation
 - Cranial nerve involvement through closure of foramina
 - Facial paralysis and deafness
 - Overgrowth of the alveolar process.

Radiographic Findings

- Increased density of many bones.
- Diffuse sclerosis of skull.

Differential Diagnosis

- Osteopetrosis
- Osteitis deformans
- Progressive diaphyseal dysplasia.

Treatment

- No treatment.

MASSIVE OSTEOLYSIS

(Vanishing Bone ; disappearing bone; phantom bone)

- Characterized by spontaneous, progressive resorption of bone with ultimate total disappearance of the bone.
- Unknown etiology but related to an active hyperemia of bone.

Clinical Features

- May or may not be painful, begins suddenly and advances rapidly until the involved bone is replaced by a thin layer of fibrous tissue surrounding a cavity.

Oral Manifestations

- Pain
- Facial asymmetry
- Pathologic fracture following minor trauma.

Histologic Features

- Typical feature is replacement of bone by connective tissue containing many thin-walled blood vessels.

Treatment and Prognosis

- No specific treatment
- Radiation or surgical resection can be used.

DISEASES OF THE TEMPOROMANDIBULAR JOINT

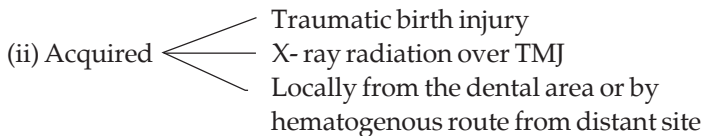
Classification

- **Developmental Disturbances of the TMJ**
 - Aplasia of the mandibular condyle
 - Hypoplasia of the mandibular condyle
 - Hyperplasia of the mandibular condyle.
- **Traumatic Disturbances of the TMJ**
 - Luxation and subluxation
 - Ankylosis
 - Injuries of the articular disk
 - Malocclusion
 - Blow /Fall
 - Yawning
 - Rheumatoid arthritis
 - Fractures of the condyle.
- **Inflammatory Disturbances of the TMJ**
 - Arthritis due to a specific infection
 - Rheumatoid arthritis
 - Osteoarthritis
 - Traumatic arthritis.
- **Neoplastic Disturbances of the TMJ**
 - Tumors may originate from the condyle, either from the bone or the articular cartilage or from the joint capsule.
 - Rarely metastatic tumors may also involve the TMJ.
- **Extra-articular Disturbances of the TMJ**
 - Impacted molar tooth on the same side as TMJ pain.
 - Sinusitis and middle ear disease.
 - Infratemporal cellulitis.

- Impingement of the coronoid process on the tendon of the temporal muscle.
- Neuritis of the 3rd division of 5th cranial nerve.
- Odontalgia.
- A foreign body in the infratemporal fossa.
- Overclosure of the mandible due to severe dental attrition.
- Costen's syndrome.
- Myofacial pain - Dysfunction syndrome

HYPOPLASIA OF THE MANDIBULAR CONDYLE

May be (i) Congenital



Clinical Features

- Facial asymmetry (severe unilateral involvement).
- Limitation of lateral excursion.
- Exaggeration of the antegonial notch.
- Mandibular midline shift during opening and closing.

Treatment

- Cartilage or bone transplants.

LUXATION AND SUBLUXATION

(Complete and Incomplete Dislocation)

Dislocation of the TMJ occurs when the head of the condyle moves anteriorly over the articular eminence into such a position that it can not be returned voluntarily to its normal position.

The following are some of the causes for luxation:

- Sudden traumatic injury resulting in fracture of condyle.
- Stretching of the external pterygoid muscle into the capsule.
- Yawning or opening the mouth too widely during extraction or removing tonsils or use of a mouth prop.

Clinical Features

- A sudden locking and immobilization of the jaws when the mouth is opened.
- Prolonged spasmodic contraction of the temporal, internal pterygoid and masseter muscles with protrusion of the jaw.
- Mouth can not be closed.
- No possibility of any movement involving mandible.

Treatment

- Reduction by an inferior and posterior pressure of the thumbs in the mandibular molar area.

ANKYLOSIS

(*Hypomobility*)

Etiology

- Abnormal intrauterine development.
- Birth injury.
- Trauma to the chin forcing the condyle against the glenoid fossa with bleeding into joint space.
- Malunion of condylar fractures.
- Injuries associated with fractures of malar - zygomatic compound.
- Loss of tissues with scarring.
- Congenital syphilis.
- Primary inflammation of the joint.
- Inflammation of the joint secondary to a local inflammation.
- Inflammation of the joint secondary to a blood stream infection.
- Metastatic malignancies.
- Inflammation secondary to radiation therapy.

Clinical Features

- Complete ankylosis → Bony fusion with no movement.
- Fibrous ankylosis → Comparatively greater motion.
- Facial deformity occurs if the injury occurs in infancy or childhood.
- Unilateral ankylosis → Chin is displaced laterally and backward on the affected side.

- Bilateral ankylosis - Receding chin and micrognathia.
- TMJ ankylosis
 - Intra-articular
 - Extra-articular
- Intra-articular ankylosis—Progressive destruction of the meniscus with flattening of the mandibular fossa, thickening of the head of the condyle and narrowing of the joint space. No movement is possible.
- Extra-articular ankylosis—Splinting of the TMJ by a fibrous or bony mass external to the joint proper movement is possible.

Radiographic Findings

- Abnormal or irregular shape of the head of the condyle and a radiopacity indicative of the dense bone filling the joint space.

Treatment

- Osteotomy or removal of a section of bone below the condyle.

COSTEN'S SYNDROME

- Costen's syndrome is the most common condition producing symptoms referable in part to the TMJ.

Clinical Features

- Impairment of hearing either continuous or intermittent
- "Stuffy" sensation in the ears, especially at meal time
- Tinnitus
- Otagia
- Dizziness
- Headache about the vertex, occiput and behind the ears
- Burning sensation in the throat, tongue and side of nose.

MYOFACIAL PAIN - DYSFUNCTION SYNDROME (MPDS)

(TMJ Pain-Dysfunction Syndrome)

- The principal factor responsible for the manifestation of this syndrome is masticatory muscle spasm which is initiated by the following factors.
- Muscular overextension—Dental restoration or prosthetic appliance which encroach intermaxillary space.

- Muscular overcontraction—Bilateral loss of post. teeth, alveolar bone resorption after wearing prosthetic appliance.
- Muscle fatigue—Chronic oral habits (Psycho-physiologic theory).

Clinical Features

- The four cardinal signs symptoms are
 - Pain usually one sided.
 - Muscle tenderness (neck of condyle, above max. tuberosity, angle of mandible and temporal crest).
 - A clicking or popping noise in the TMJ.
 - Limitation of jaw motion with deviation on opening.
- Occlusal disharmony.
- Female preponderance between 20 - 40 years.
- Gradual onset.
- Usually self-limiting.
- The two typical negative disease characteristic are
 - Absence of clinical, roentgenographic or biochemical evidence of organic changes in the joint itself.
 - Lack of tenderness in the joint when palpated through the external auditory meatus.
- Specific muscles affected are
 - Lateral pterygoid
 - Masseter
 - Temporalis
 - Medial pterygoid
 - Cervical, scalp and facial muscle.

Treatment

- The following are the principles of management of MPDS.
 - Reassurance
 - Habit management and physical therapy.
 - Occlusal appliance.
 - Analgesia (tranquilizers muscle relaxants).
- Treatment is conservative.
- Surgery, drug injection into joint, extensive occlusal equilibration should be avoided in the early phases of treatment.
- Myotherapeutic exercises.
- Correction of faulty restoration or appliances.

Diseases of the Skin

HEREDITARY HYPOHIDROTIC ECTODERMAL DYSPLASIA

(Christ - Siemens- Touraine syndrome)

- Specific syndrome characterized by congenital dysplasia of one (or) more ectodermal structures and their accessory appendages → manifested by Hypohidrosis, Hypotrichosis, Hypodontia.

Etiology

- X- Linked recessive Mendelian characteristics.
- Males more affected than females. Autosomal dominant or recessive characteristic.

Clinical Features

- Soft, smooth, thin, dry skin with partial or complete absence of sweat glands. Persons can not perspire which leads to hyperpyrexia and intolerance to warmer temperatures.
- Children develop this as their first symptom.
- Sebaceous glands, hair follicles are often defective (or) absent.
- Hairs of the scalp, eyebrows are fine and scanty.
- Bridge of nose is depressed; supraorbital ridges, frontal bosses are pronounced and the lips protuberant.

- Facial appearance is such that they resemble each other and are mistaken for siblings.

Oral Manifestations

- Anodontia (or) oligodontia, complete or partial absence of teeth.
- When some teeth are present, they are truncated (or) cone shaped.
- Absence of teeth, leads to impaired or reduced development of the alveolar process and hence reduced vertical dimension, resulting in protuberant lips.
- High arch palate, cleft palate.
- Hypoplastic intraoral accessory gland results in xerostomia. Hence protuberant lips which may be dry, cracked with pseudorhagades formation.

Treatment

- Dentures → Periodical reconstruction as the jaws grow.

CHONDROECTODERMAL DYSPLASIA

(Ellis - van Creveld syndrome)

- Similar to hereditary anhidrotic ectodermal dysplasia.
- Not classified as a dermatologic disease.

Etiology

- Autosomal recessive characteristic.

Clinical Features

- Ectodermal disturbances of nails, teeth, chondrodysplasia, polydactyly, and sometimes congenital heart diseases.
- Nails → Hypoplastic with marked koilonychia.
- Bilateral cases of hands and feet - polydactyly.

Oral Manifestations

- Fusion of middle portion of upper lip to maxillary gingival margin → elimination of normal mucolateral sulcus.

Treatment

- No treatment.

LICHEN PLANUS

- One of the most common dermatologic diseases to manifest in the oral cavity.

Etiology

- Unknown
- Nervous, high string person
- Persons of emotional upset, overwork, anxiety, mental strain
- Traumatism
- Malnutrition
- Infection
- Immune factor (complement, Immunoglobulins are seen at dermal-epidermal junction.
(vascular hypertension + diabetes mellitus + lichen planus)-
Grinspan's syndrome.

Clinical Features

- Appear as flat - topped, angular, small papules one millimeters in diameter.
- May be discrete (or) coalesce into larger plaques covered by a glistening, scale.
- Papules are sharply demarcated from the surrounding skin.
- Early lesions are red. Late are violaceous, reddish to purple. Later dirty brown color.
- Centre of the papule is slightly umbilicated.
- Its surface is covered by characteristic, fine grayish white lines called 'Wickham's striae'.
- Bilaterally symmetrical.

Sites

- Flexor surfaces of wrist, arms, inner aspect of knees, thighs, sacral area.

Primary Symptom

- Intolerable pruritus.

Sex Predilection

- Females are affected more than males.

Oral Manifestations

- Female predilection.

Age

- Seen in 40 - 70 years.

Appearance

- Radiating, white, velvety, thread-like papules, in a linear, annular, retiform arrangement forming typical lacy patches, or rings and streaks over the buccal mucosa.
- White elevated dot is present at the intersection of white lines (Wickham's striae).

Site

- Buccal Mucosa - 80%
- Tongue - 65%
- Lips - 20%
- Gingiva , Floor of the mouth palate - 10%.

Types

- Bullous form
- Erosive form
- Atrophic
- Hypertrophic.

Erosive Forms

- Eroded ulcers
 - Irregular in size, appear, raw, painful areas
 - Radiating striae are still noted even though erosion is present.

Atrophic

- Smooth red, poorly defined areas, not always with peripheral striae.

Hypertrophic

- Well-circumscribed, elevated white lesion, resembling leukoplakia.

Oral lesions

- Occur weeks (or) months before skin lesions.

Histologic Features

Hyperparakeratosis (or) Hyperorthokeratosis

- With granular layer thickening and acanthosis with intracellular edema of spinous cells.
- Development of 'saw-tooth' rete pegs.

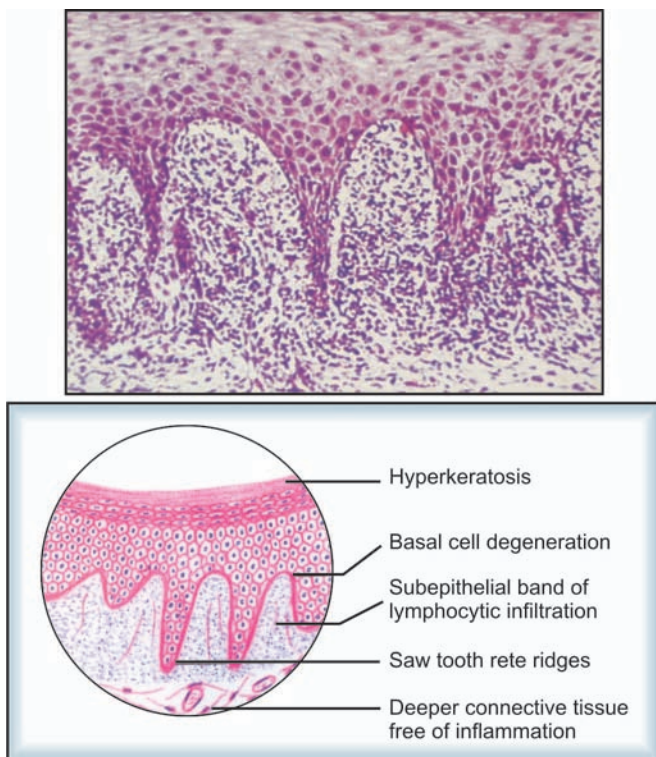


Fig. 16.1: Lichen planus

Liquefaction of Basal Cell Layer

- With appearance of a thin band of eosinophilic coagulum in its place.
- Infiltration of lymphocytes in the subepithelial layer of connective tissue.
- Colloid bodies also called Civatte, hyaline (or) fibrillar bodies present in the basal, spinous cells layers.
- Appear as red, eosinophilic, globules, representing degenerated epithelial cells (or) phagocytosed epithelial cell remnants.

Electron Microscopic Features

- Irregular nuclear membrane of cells.
- Increased thickening, granularity to tonofibrils.

Treatment

- Vitamin therapy.
- Corticosteroids → Intralesional.

PSORIASIS

- Chronic inflammatory dermatologic disease
- Rarely in oral mucous membrane.

Clinical Features

- Small, sharply, delineated papules, covered by delicate silvery scale, resembling thin layer of mica.
- If deep scales are removed, tiny bleeding points are enclosed → characteristic feature termed. 'Auspitz Sign'.
- Painless, seldom pruritic lesions.
- Roughly symmetrical, elevated large plaques usually on extensor surfaces of the extremities, particularly the elbows, knees, scalp, back and chest.
- More severe in winter, less severe in summer, as a result of increased exposure to ultraviolet light.

Oral Manifestations

- Described as gray (or) yellow white plaques; as silvery, white, scaly lesions with an erythematous base.

- As multiple papular eruptions which may be ulcerated, or as small, papillary, elevated lesions.

Histologic Features

- Uniform parakeratosis.
- Absence of stratum granulosum.
- Elongated, clubbed retepegs.
- Epithelium is thinned and from these points, the bleeding occurs when scales are peeled off.
- Intraepithelial abscesses (Monro's abscess) are a common finding.
- Mild histiocytic, lymphocytic infiltration particularly perivascular, periadenexal in location.
- Turn over time for psoriatic skin lesions 3-4 days.

Treatment

- Topical corticosteroids, tars, drugs such as dihydroxyanthralin, retinoic acid, iodochlorohydroxygin.

PYTRIASIS ROSEA

- Acute condition of unknown etiology.

Clinical Features

- Superficial light red macules (or) papules - generalized over most of the skin surfaces → Trunks, Thigh, Groin.
- Generalized outbreak is preceded by the appearance of 'Primary lesion' (or) 'Herald spot', seven to ten days previously.
- Size of the lesion (3-4 cm in diameter).
- Lesions are ovoid, covered by a thin silvery scale.
- Symptoms → Mild itching, headache, low-grade fever, cervical lymphadenopathy.
- Duration → Stays for 3-6 weeks.
- Age → Seen in young adults.
- Cause → Unknown virus, nervous strain, emotional upsets were considered.

Oral Manifestations

- Site → Buccal mucosa, tongue, palate.
- Lesion → Appears as erythematous macules with (or) without central area of greyish desquamation.
- Show raised border, 1-2 cm diameter.
- Asymptomatic.

Histologic Features

- Slight acanthosis, focal parakeratosis with microvesiculation or sprinkling of leukocytes within the epithelium.
- Edema, hyperemia, perivascular infiltration of histiocytes lymphocytes, plasma cells.

Treatment

- Self limiting.

Erythema Multiforme

- Ulcerative, blistering, mucocutaneous condition of unknown pathology.
- Immunologically mediated process.
- Herpes simplex, mycoplasma pneumoniae, exposure to drugs trigger immunologic derangement that produces the disease.
- Molecular biology - showed herpes simplex DNA in patients with recurrent erythema multiforme.

Clinical Features

- Acute onset, with ulcerations of the oral mucosa.
- Most severe form → Ulceration and diffuse sloughing of skin and mucous membrane are seen. (Toxic epidermal Necrolysis (or) Lyell's disease).
- Age → Young adults (20 or 30 years).
- Sex → Male predilection.
- Prodromal symptoms → Fever, malaise, headache, cough, sore-throat, one week before onset.
- Most cases are self - limiting (in 2-6 weeks).

- **Skin lesions** → May be of variety of appearances.
- **Early lesions** → Flat, round, dusky red → Slightly elevated and evolve into bullae with necrotic centers.
- The lesions appear as concentric circular erythematous rings resembling target (or) bull's eye lesion.
- Oral lesion begins as erythematous patches that undergo epithelial necrosis and evolve into, large, shallow erosions and ulcerations with irregular borders.
- Hemorrhagic crusting of vermillion zone of lips.
- Lesions of oral cavity emerge quickly and are uncomfortable.

Common Sites of Involvement

- Lips, labial mucosa, buccal mucosa, tongue, floor of the mouth, soft palate.

ERYTHEMA MULTIFORME MAJOR

(Stevens - Johnson syndrome)

- Ocular (or) genital mucosal are affected in conjunction, with oral, skin lesions. With severe ocular involvement, scarring may occur. (Symblepharon).
- Toxic epidermal necrolysis.
- Most severe form of erythema multiforme.
- Triggered by drug exposure.
- Age → Older people.
- Sex → Female predilection.
- Diffuse sloughing of skin, mucosal surfaces.
- Cutaneous process resolves in 2-4 weeks.
- Oral lesions may take a longer time to heal.

Histopathology

- Intra-epithelial vesiculations with necrotic basal keratinocytes.
- Mixed, inflammatory cell infiltrate is seen.

Treatment

- Use of systemic corticosteroids → at early stages.
- Avoid the etiological factor.

- Erythema multiforme minor → symptomatic treatment, bland mouth rinses.
- Erythema multiforme major → topical corticosteroids along with antifungals.

EPIDERMOLYSIS BULLOSA

- Heterogenous group of inherited blistering mucocutaneous disorders.
- It has a specific defect in the attachment mechanisms of epithelial cells between each other (or) underlying connective tissue.
- Three broad categories based on cellular cohesion → defect mechanism.
 - Simplex
 - a. Generalized form
 - b. Localized form (Weber-Cockayne syndrome)
 - Junctional
 - a. Dominant
 - b. Recessive
 - Dystrophic
 - Acquisita (Acquired).

Simplex: *Generalized form* → It is inherited as an autosomal dominant characteristic, manifests at birth (or) shortly after.

Characterized by vesicle (or) bulla formation chiefly on hands and the feet. When the blisters heal, in 2-10 days, no resultant scarring (or) permanent pigmentation is seen.

Disease appears to improve at puberty.

Localized form → Occur early in childhood, and is recurrent. Occur on hands, feet. No scarring is seen.

Oral Manifestation

- Rarely then occur in the oral cavity.

Histology

Generalized Form

- Destruction of basal and suprabasal cells so that some nuclei are present on the floor of the blister.

- Individual cells show dissolution of organelles and tonofibrils, with displacement of nucleus to upper end of the cell.
- Localized → Bullae are intraepidermal and suprabasal in location.

DYSTROPHIC

(Dominant)

Clinical Features

- Onset → Infancy (or) delayed until puberty.
- Sites → Blisters are seen on ankles, knees, elbows, feet, head.
- Healing results in scarring, nails are thick and dystrophic, milia are present.
- Palmar → Plantar keratoderma is seen along with ichthyosis, hypertrichosis.

Oral Manifestations

- Teeth are unaffected.
- Oral milia were described.

Histologic Features

- Bullae develop as a result of separation through the very thin, irregular PAS positive basement membrane which becomes divided.

DYSTROPHIC

(Recessive)

- Classic form of disease.
- Seen at birth.
- Bullae are seen at sites of trauma, friction or pressure; feet, buttocks, scapulae, fingers.
- Blister contains fluid which is blood - tinged.
- Bullae rupture on leaving a raw surface that is painful.
- Have a positive Nikolsky's sign.
- Bullae show healing with scar, milia, pigmentation.
- Scar results in a functional club-like fists.

Oral Manifestations

- Oral bullae are common. They may be preceded by appearance of white spots (or) patches on the oral mucous membrane.
- Bullae are initiated by any operative procedure in oral cavity.
- Bullae are painful, when they rupture. Scar formation results with hoarseness and dysphagia; esophageal strictures.

Dental Defects

- Rudimentary teeth, congenitally absent teeth, hypoplastic teeth, crowns denuded of enamel.

Histology

- Fragments of basement membrane adhere to dermis. The basal cells are normal.
- Pre-elastic and oxytalan fibres are increased in number.

JUNCTIONAL TYPE

- **Lethal type** (or) severe form of dystrophic recessive form.

Clinical Features

- Onset at birth.
- Absence of scarring, milia (or) pigmentation.
- Death within 3 months of age.

Oral Manifestations

- Produces serious feeding problems.
- Disturbances of enamel, dentin of deciduous teeth are seen.

Histology

- Identical to dystrophic - recessive type.

ACQUISITA TYPE

- Rare form with no evidence of hereditary transmission.

Onset

- In adult life. Bullae is chiefly symptomatic.
- Simplex form, requires little treatment.

- Antibiotics are used for secondary infection control.
- Lethal form, terminates fatally.

PEMPHIGUS

- It is a serious, chronic inflammatory disease of the skin, that shows vesicles (or) bullae, small or large fluid - filled blister which develop in cycles.

Etiology

- Autoimmune in origin.

Types

- *Pemphigus vulgaris.*
- *Pemphigus vegetans.*
- *Pemphigus foliaceus.*
- *Pemphigus erythematosus.*

General Features

- Initially the lesion starts as a vesicle (or) bulla and then ruptures leaving a raw eroded surface.
- The primary site of the lesions are the oral mucosa, and also occurs on the trunk and other regions.

(a) Pemphigus Vulgaris

- It is the most common type of pemphigus.
- It is common in the oral mucosa also.
- It is seen as a vesicle (or) a bulla that is usually few millimeters to centimeters in diameter.
- It covers extensive areas of the skin.
- The fluid in the blister is thin and watery and soon it becomes purulent (or) sanguineous.
- Then the vesicle ruptures leaving a raw erroded surfaces that resembles identical focal areas, that are formed by peeling of epithelium due to oblique pressure without formation of the vesicle.

- The peeling of epithelium, due to rubbing of the unaffected skin is known as 'Nikolsky's sign'
- This sign is positive in pemphigus.
- Recovery may take months to years for the disease (or) it may terminate in death.

(b) Pemphigus Vegetans

- These also start as vesicles (or) bullae. Later they rupture leaving a raw eroded surface.
- 'Fungoid' like masses (or) vegetations start growing on the raw surface.
- These masses are covered by a purulent exudate.
- These lesions are surrounded by an inflammatory border.
- They seem to arise from *Pemphigus vulgaris* (or) vegetans and terminate within months and exhibit remissions.
- Common on the nose, mouth.

(c) Pemphigus Foliaceus

- It arises from *Pemphigus vulgaris* (or) vegetans.
- It arises as a vesicle (or) bullae and ruptures leaving a raw eroded surface.
- This surface dries forming scales, which resembles 'exfoliative dermatitis' (or) 'eczema'.

It is rarely seen in the oral mucosa, and is a mild form of pemphigus.

(d) Pemphigus (Brazilian type)

- This type of pemphigus foliaceus is common in tropical regions such as Brazil.

(e) Pemphigus Erythematosis

- Here vesicles are seen, which rupture and are concomitant with crusted patches formation, that resembles 'seborrheic dermatitis'. It is not seen in the oral mucosa. It terminates in pemphigus vulgaris (or) vegetans.
- It is usually associated with malaise and fever.

Oral Manifestations

- Vesicle and bullae are commonly seen in the mucosa of oral cavity in pemphigus vulgaris and vegetans type.
- They are not usually seen, as they don't stay for a longer time and ruptures soon.
- Nikolsky's sign is seen in the oral mucosa.

Cause for this Sign

- Prevesicular edema which ruptures the dermal - epidermal junction.
- No intraoral site is immune to pemphigus.
- The sites bleed easily and renders difficulty and pain while eating.
- Surface material contains eosinophils.

Histopathology

- Distinctive suprabasilar split (or) intra-epithelial cleft.
- Is seen in the epithelium.
- Prevesicular edema disrupts the dermal-epidermal junctions, as well as intercellular bridges.
- Results in acantholysis.
- These shed spinous cells becomes rounded and are often clumps, and are eosinophilic in nature.
- This leads to formation of 'TZANCK' cells.
- Exfoliative cytology that is done when the fresh vesicle ruptures, shows these 'TZANCK Cells' and this test used to demonstrate these cells is called 'TZANCK test'.

Confirmatory Tests

- Direct immunofluorescence test detects the presence of Ig G, Ig A, Ig M antibodies and C₃.

Treatment

- Corticosteroids and antibiotic therapy for secondary infection both systemic as well as topical therapy is important.

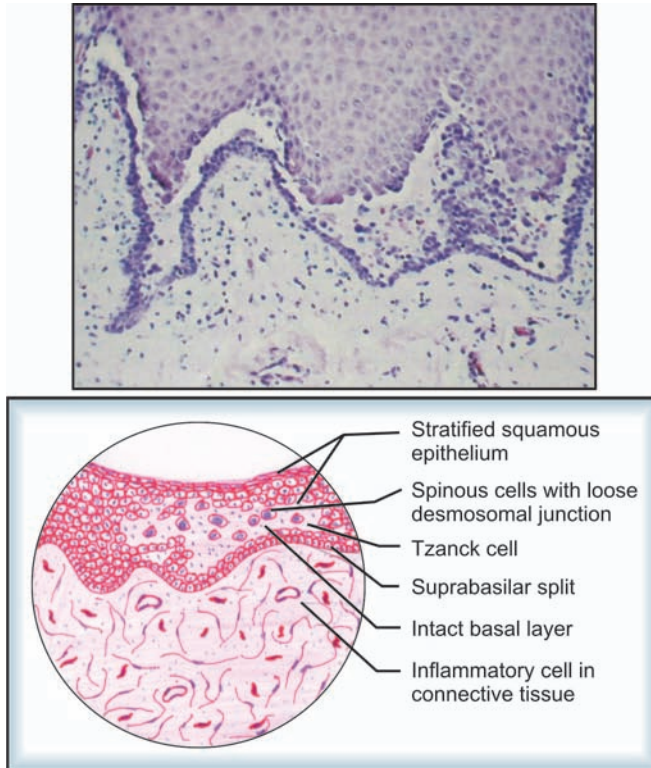


Fig. 16.2: Pemphigus

LUPUS ERYTHEMATOUS

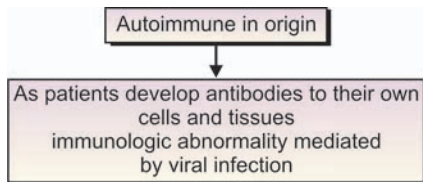
- Common skin disease of unknown etiology.

Common Types

Two types are common

- Systemic lupus erythematosus.
- Lupus erythematosus - discoid.
- Both have certain similarities in lab findings.
- Presence of antinuclear antibodies, and elevated ESR.

Etiology



Clinical Features

Systemic Type

- *Age:* 30 to 40 years.
- *Sex:* 8:1 ratio with a female predilection.
- Cutaneous lesions show erythematous patches on the face, coalesce to form symmetrical patterns over the cheek. Itching and burning sensation with hyperpigmentation. Severity intensifies on exposure to sunlight. Associated with rheumatoid arthritis, kidney and heart diseases.

Discoid Type

- *Age:* 30-40 years.
- *Sex:* Female predilection.
- *Site:* Oral mucosa, chest, back.
- *Cutaneous lesions:* Elevated red or purple macules covered by grey or yellow adherent scales.
- Periphery is pink or red, sometimes show scarring.
- Sometimes assumes a 'butterfly' distribution.

Oral Manifestations

- Discoid lesions begin as erythematous areas, slightly elevated without induration.
- Pain and bleeding may be present.
- Fine white striae radiating from margins.

Site

- Buccal mucosa, lip, tongue.
- Systemic lesions → begin as discoid lesions with hyperemia, oedema and xerostomia → end fatally.

Histology

- Hyperkeratosis with atrophy of rete pegs.
- Degeneration of basal layer.
- Infiltration of lymphocytes.
- Hyalinization.
- Edema beneath epithelium.

Systemic Lesions

- Shows degenerative features prominently lymphocytic infiltration less severely.

Discoid Lesions

- Shows hyperorthokeratosis with epithelial atrophy; acanthosis and keratin plugging is seen.

Confirmatory tests → Presence of IgG, IgA, IgM at the dermal, epidermal junction by direct immunofluorescence test.

Histology

- Thickening of basement membrane is demonstrated as a PAS-positive acellular band.
- Diffuse infiltration of lymphocytes, plasma cells and polymorphonuclear leukocytes are in superficial and deep connective tissue.
- Focal perivascular collection of lymphocytes are found along with degeneration and disintegration of collagen.

Systemic type → Have the same characteristic features with exception of absence of keratinization.

Lab Findings

- A specific test was established with discovery of “L.E” cell inclusion phenomenon.
- **Test consists** of, essentially in addition of blood serum from a person under suspicion to the buffy coat of normal blood.
- If patient is suffering from systemic Lupus erythematosus, typical ‘L.E’ cells develop.

- This cell consists of a rosette of neutrophils surrounding a pale nuclear mass apparently derived from a lymphocyte.
- The basis appears to lie in the gammaglobulin of the serum from the patient.
- Only in rare occasions is the L.E cell found in discoid Lupus erythematosus.
- Antinuclear antibodies are demonstrated in both forms of the disease. (Common in Systemic Lupus).

Treatment

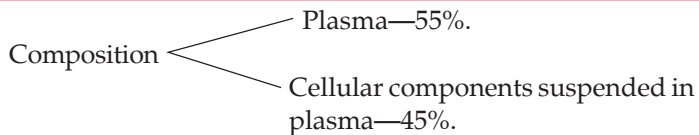
- Terminated fatally most of the times.
- Corticosteroid and/or Antimalarial drugs.
- Discoid form may be slowly progressive over a period of many years.

Diseases of Blood and Blood Forming Organs

INTRODUCTION

Physiology

- Blood is described as a connective tissue providing one of the means of communication between the cells of different parts of the body and the external environment.
- Constitutes about 7 percent of body weight.

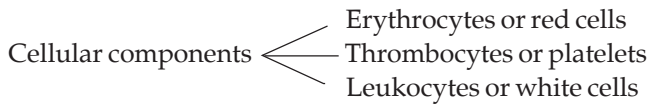


- Plasma is a straw colored fluid.
- Serum is plasma without fibrinogen.

Constituents

- Plasma proteins
- Albumin
- Globulin
- Fibrinogen
- Clotting factors

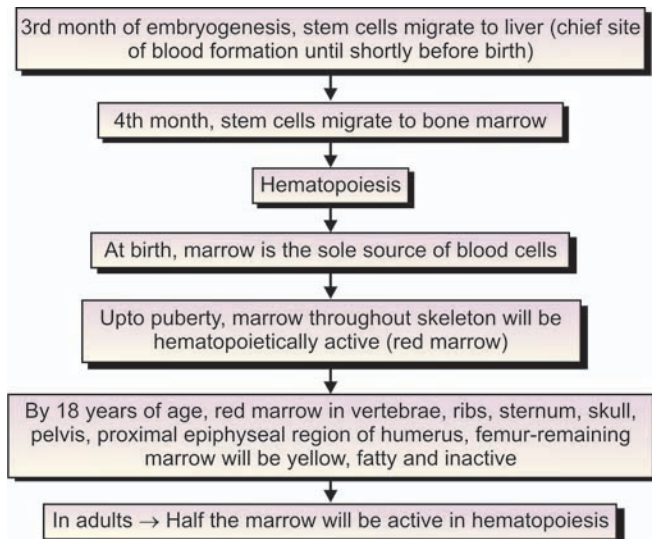
- Inorganic salts (Mineral salts)
- Nutrient materials
- Organic waste materials
- Hormones, enzymes, antibodies
- Gases—Oxygen, carbon dioxide



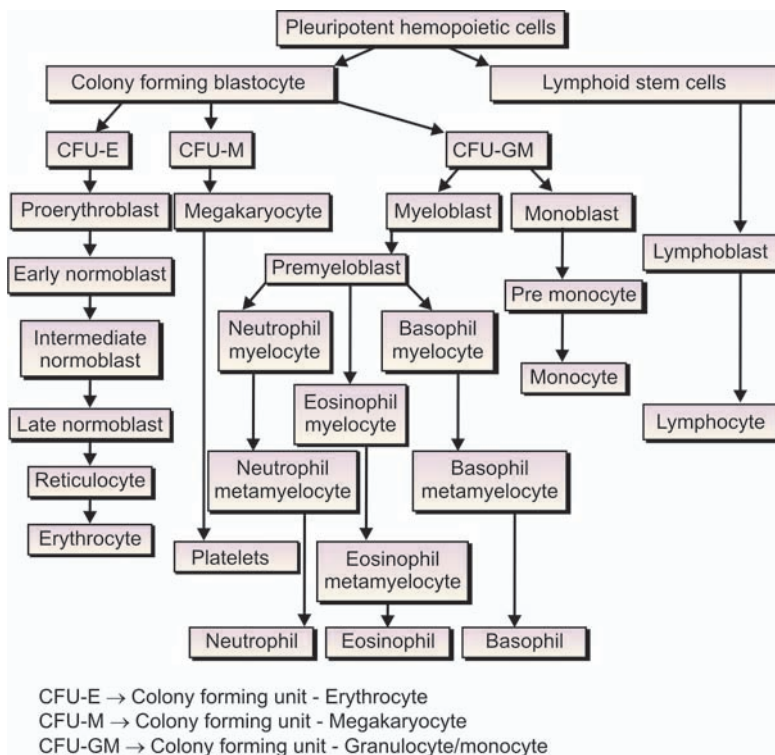
Development, Origin, Differentiation of Blood Cells

- First appears during 3rd week of fetal embryonic development in yolk sac.
- Originate from stem cells (hemocytoblasts).
- Hematopoietic stem cell originates in mesoderm of intraembryonic aorta/gonad (or) within a small subset of yolk sac derived cells.

Development of Blood Cells



Origin of Blood Cells



Erythrocytes

- Circular biconcave non-nucleated.
- 7 μ diameter.
- Carries oxygen to peripheral tissues.

Leukocytes

Types

Granulocytes

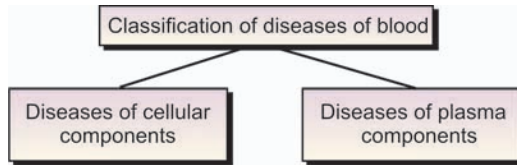
- Neutrophils
- Eosinophils
- Basophils

Agranulocytes

- Monocytes,
- Lymphocytes

Function

- Defend body against microbes and other foreign materials



Diseases of Cellular Components

- RBC'S—Anemia, polycythemia.
- WBC'S—Leukopenia, leukocytosis, leukemia.
- Platelets—Thrombocytopenia, thrombocyasthenia.

Diseases of Plasma Components

Clotting Factors Deficiency

- Hemophilia
- von Willebrand's disease
- Factor XIII deficiency.

Plasma Proteins

- Afibrinogenemia
- Dysfibrinogenemia
- Cryoglobulinemia.

DISEASES OF RBC'S

ANEMIAS

Definition

Anemia is defined as an abnormal reduction in the number of circulating red blood cells, the quantity of hemoglobin and the volume of packed red cells in a given unit of blood.

Classification of Anemias

Etiological Classification of the Anemias

- Loss of blood.
 - Acute posthemorrhagic anemia.
 - Chronic posthemorrhagic anemia.
- Excessive destruction of red corpuscles
 - Extracorpuscular causes.
 1. Antibodies
 2. Infection (malaria, etc.)
 3. Splenic sequestration and destruction
 4. Associated disease states, e.g. lymphoma
 5. Drugs, chemicals, and physical agents
 6. Trauma to RBC.
 - Intracorpuscular hemolytic disease
 1. Hereditary
 2. Disorders of glycolysis
 3. Faulty synthesis or maintenance of reduced glutathione
 4. Qualitative or quantitative abnormalities in synthesis of globin
 5. Abnormalities of RBC membrane
 6. Erythropoietic porphyria
- Acquired
 - Paroxysmal nocturnal hemoglobinuria.
 - Lead poisoning.
- Impaired blood production resulting from deficiency of substances essential for erythropoiesis
 - Iron deficiency
- Experimentally; also copper and cobalt deficiencies.
 - Deficiency of various B vitamins

Clinically, B₁₂ and folic acid deficiencies (pernicious anemia and related macrocytic, megaloblastic anemias); pyridoxine - responsive anemia.

Experimentally, pyridoxine and niacine deficiencies; possibly also riboflavin, pantothenic acid and thiamine deficiencies.

 - Protein deficiency.
 - Possibly ascorbic acid deficiency.
- Inadequate production of mature erythrocytes
 - Deficiency of erythroblasts

1. Atrophy of bone marrow: aplastic anemia
2. Chemical or physical agents
3. Hereditary
4. Idiopathic.
- Isolated erythroblastopenia ('pure red cell aplasia')
 1. Thymoma
 2. Chemical
 3. Antibodies.
- Infiltration of bone marrow
 1. Leukemia, lymphomas
 2. Multiple myeloma
 3. Carcinoma, sarcoma
 4. Myelofibrosis.
- Endocrine abnormality
 1. Myxedema
 2. Addisonian adrenal insufficiency
 3. Pituitary insufficiency
 4. Sometimes, hyperthyroidism.
- Chronic renal disease
- Chronic inflammatory disease.
 1. Infections
 2. Noninfectious diseases, including granulomatous and collagen diseases.
- Cirrhosis of liver.

MORPHOLOGIC CLASSIFICATION OF THE ANEMIAS

<i>Types of anemia</i>	<i>Description</i>
1. Macrocytic	Increased normal
2. Normocytic	Reduction normal normal
3. Simple microcytic	Reduced normal
4. Hypochromic microcytic	Reduced reduced

MOST COMMON CAUSES

MCV; increased MCH; MCH Cone	Lack of erythrocyte-maturing factors (‘extrinsic’ and ‘intrinsic’ factors)
Only in RBC number; MCV; normal MCH; MCH Cone	Hemorrhage; hemolysis; lack of blood formation; dilution of blood with fluid
MCV; reduced MCH; MCH Cone	Associated with infections and inflammatory diseases
MCV; reduced MCH; MCH Cone	Iron deficiency

MCV = mean corpuscular volume (Volume/RBC).

MCH = mean corpuscular hemoglobin (Hb/RBC).

MCH Cone = mean corpuscular hemoglobin concentration (Hb/Vol).

KINETIC CLASSIFICATION OF ANEMIA

- Impaired erythrocyte production (Reticulocyte production index less than 2)
 - Hypoproliferative
 - Iron - deficient erythropoiesis
 - Iron deficiency
 - Anemia of chronic disorders
 - Erythropoietin deficiency
 - Renal disease
 - Endocrine deficiencies
 - Hypoplastic anemia
 - Aplastic anemia
 - Pure red cell aplasia
 - Infiltration
 - Leukemia
 - Metastatic carcinoma
 - Myelofibrosis
 - Ineffective
 - Megaloblastic
 - Vitamin B₁₂ deficiency
 - Folate deficiency
 - Other causes

- Microcytic
Thalassemia
Certain sideroblastic anemia
- Normocytic
- Increased erythrocyte production (Reticulocyte production index greater than 3)
 - Hemolytic anemia
 - a. Hereditary
 - b. Acquired
 - c. Treated nutritional anemia

IRON DEFICIENCY ANEMIA

Most common type characterized by microcytic hypochromic red blood cells [i.e. RBC'S are smaller in size and have decreased Hb].

Etiology

- Chronic blood loss—excessive menstruation, postmenopausal uterine bleeding.
- Increased requirements of iron in pregnancy, lactation, infancy, childhood and adolescence.
- Increased dietary intake.
- Decreased absorption in partial or total gastrectomy and chlorhydria.

Plummer-Vinson syndrome—manifestation of iron deficiency anemia. Originally described as “Hysterical dysphagia” Predisposes to cancer of upper alimentary tract (Premalignant condition).

Clinical Features

- Women > Men—women have more predilection than men.
- Seen in 4th - 5th decade of life.
- Fissures at angle of mouth.
- Atrophy of tongue papillae causes smooth red painful tongue.
- Oral, esophageal mucous membrane shows pallor, atrophy and loss of keratinization.
- Koilonychia (spoon-shaped nails).
- Esophageal stricture/web heads to dysphagia.

- Lemon tinted pallor of skin.
- Decreased RBC count.
- Serum “Fe” is low.
- Microcytic hypochromic RBC’S in peripheral smear.
- Exfoliative cytology of squamous epithelial cells from tongue shows deficiency of keratinized cells, decreased diameter of cells and increase in size of nucleus increased nucleoli.

Treatment

- Iron therapy, high protein diet.

MEGALOBLASTIC ANEMIA

Causes

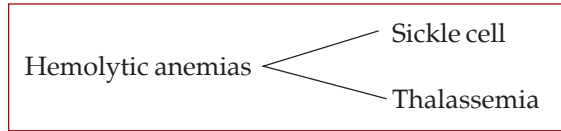
- Vitamin B₁₂ deficiency, folate deficiency.
- Vitamin B₁₂ deficiency occurs in, (1) inadequate dietary intake, (2) malabsorption (as in lack of intrinsic factor in gastric juice necessary for B₁₂ absorption).
- Pernicious anemia is due to intrinsic factor deficiency. Intrinsic factor is responsible for vitamin B₁₂ absorption. Lack of intrinsic factor in gastric juice causes vitamin B₁₂ deficiency.

Clinical Features

- Weakness.
- Numbness of extremities, sensory disturbances.
- “Beefy - red” tongue.
- Painful burning sensation in tongue.
- Glossitis (Inflamed tongue), glossodynia (painful tongue), glossopyrosis (burning tongue).
- Bald tongue - “Hunter’s glossitis” or “Moeller’s glossitis.
- Rarely-ulcers are seen.
- RBC’S Peripheral and smear shows macrocytic, stippled RBC’S, Howell - Jolly bodies, large polymorphonuclear leukocytes with large polylobed nuclei.
- Bone marrow shows megaloblasts.

Treatment

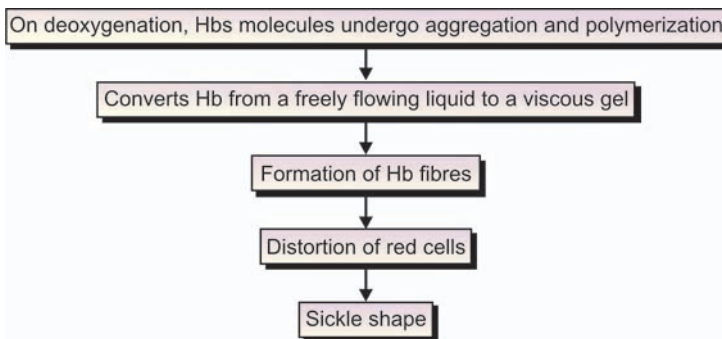
- Vitamin B₁₂ administration



SICKLE CELL ANEMIA

- Hereditary type of hemolytic anemia.
- Etiology: HbA (Adult Hb) is replaced by Hbs valine replaces glutamic acid in the β -globin chain, leading to formation of Hemoglobin S (Hbs).

Pathogenesis



Clinical Features

- Tissue anoxia due to vascular occlusion by sickled cells
- Pain in joints, limbs, abdomen.
- Radiographically shows osteoporosis, loss of trabeculation of jaw bones (especially alveolar bone).
- Hair on end/crew - cut appearance on X-ray skull.

Treatment

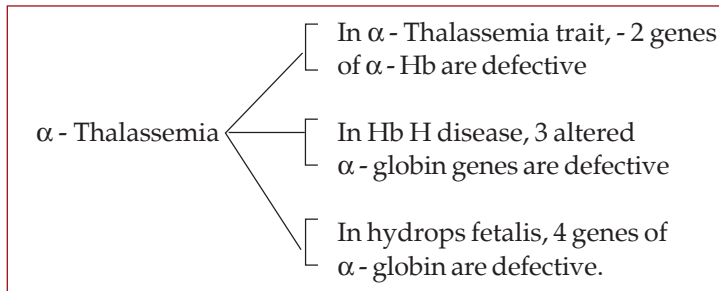
- Blood transfusion.

THALASSEMIA

- Hereditary type of hemolytic anemia.
- Decreased synthesis of α or β -globin- chain of Hb is seen.

Types

- α - Thalassemia – decreased α -globin chain synthesis.
- β - Thalassemia – decreased β - globin chain synthesis.
- Thalassemic major - severe form of disease.
- Thalassemia minor - mild form.
- Inheritance of 2 defective genes that code for β - globin chain of Hb causes β -thalassemia major. If only one defective gene for β -globin molecule is inherited, it causes β -thalassemia minor. Causes β -thalassemia major



Clinical Features

- Hepatosplenomegaly
- Painless enlargement of maxilla and mandible causing a “chipmunk” facies.
- Face shows mongoloid features.
- Depressed nasal bridge.
- Prominent cheek bones.
- Mongoloid slanting eyes
- Prominent premaxilla
- Flaring of maxillary anterior teeth
- Peripheral smear shows
 - Microcytic hypochromic red cells, poikilocytosis, anisocytosis.
 - In Target cells centre of RBC shows condensation of hemoglobin. Nucleated RBC’s.

- WBC Count - increased.
- Increased bilirubin due to excessive hemolysis.
- Bone marrow hyperplasia.

Radiological Features

- Skull shows “hair on end” / “crew - cut” appearance. Intraoral X-ray may represent mild osteoporosis. Coarsening and blurring of trabeculae giving a “salt pepper” appearance.

Treatment

- Blood transfusion.

APLASTIC ANEMIA

- Failure of hematopoietic precursor cells in the bone marrow to produce adequate numbers of all types of blood cells.

Types

Primary—Unknown etiology

Secondary—Due to drugs (chloramphenicol), chemicals, viruses like non-A, non-B, non-C and hepatitis viruses.

- Characterized by pancytopenia, i.e. due to lack of bone marrow activity there is in RBC, WBC, platelet count.

Clinical Features

- Symptoms of anemia—fatigue, tachycardia, weakness.
- Bleeding tendency (due to thrombocytopenia).
- Infections (Due to leukopenia).
- Oral mucosal petechiae, purpura.
- Gingival hemorrhage.
- Ulcers—in mucosa due to leukopenia.
- Normocytic—Normochromic anemia
- Bone marrow—Acellular, hyperplastic.

Treatment

- Immunosuppressive drugs, bone marrow transplantation.

POLYCYTHEMIA

- Abnormal increase in number of red blood cells with an increased hemoglobin level.

Types

- Relative polycythemia – apparent increase in the **RBC'S** due to hemoconcentration secondary to fluid loss.
- Primary polycythemia–is idiopathic in nature
- Secondary–causes bone marrow anoxia, production of an polycythemia erythropoietic stimulating factor.

Clinical Features

- Occurs in older adults.
- Ruddy complexion of skin (diffuse reddening).
- Generalized pruritis.
- Splenomegaly.
- Peptic ulcers.
- Oral mucosa - deep purplish red.
- Gingival hemorrhage, submucosal petechiae .
- Increased in **RBC** count, increased in **Hb**.
- Leukocytosis, thrombocytosis.

Treatment

- Phlebotomy
- Myelosuppressive therapy
- Inhibitors of platelet production like anagrelide hydrochloride-administered.
- Radioactive isotope of phosphorus P^{32} .

DISEASES OF WBC

Classification

Reduction in Number of WBC

- Leukopenia
- Agranulocytosis
- Cyclic neutropenia

Increase in Number of WBC

- Leukemia
- Leukocytosis.

LEUKOPENIA

- Decrease in number of WBC'S in peripheral blood stream.

Causes

- Bacterial viral, protozoal infections.
- Hemopoietic disorders, e.g. Aplastic Anemia.
- Chemicals.
- Physical agents, e.g. radiation.
- Oral mucosa—ulcers.

AGRANULOCYTOSIS

- Cells of granulocytic series, particularly neutrophils are absent.
- Idiopathic, drug induced, congenital (Kostmann's syndrome).
- High fever, chills, sore throat, infection of GIT, skin, respiratory tract.
- Necrotizing ulcers—Buccal mucosa, tongue, palate, gingiva.
- Gingival hemorrhage.
- Excessive salivation.
- WBC count less than 2000 cells /cu mm of blood.
- Absence of PMN'S and other granulocytes.

Treatment

- Withdraw usage of drugs that induced the agranulocytosis.
- Control infection with antibiotics.

CYCLIC NEUTROPENIA

(Periodic Neutropenia, Periodic Agranulocytosis)

- Idiopathic hematologic disorder characterized by periodic or cyclic reductions in circulating polymorphonuclear neutrophilic leukocytes.
- Accompanied by clinical manifestations which regress spontaneously, but recur subsequently in a rhythmic pattern.

- Defect in the hemopoietic stem cells in the bone marrow causes cyclic neutropenia.
- Symptoms begin in childhood.
- Signs/symptoms occur in uniformly spaced episodes, usually a 21 - day cycle.
- Recurrent fever, anorexia, lymphadenopathy, pharyngitis.
- Oral ulcers, gingiva severely affected.
- Periodontal bone loss, gingival recession, mobility of teeth.
- Neutrophil count less than $500/\text{mm}^3$ for 3 - 5 days during each of three successive cycles.

Treatment

- Antibiotics
- Corticosteroids
- Administration of granulocyte colony stimulating factors (G-CSF)
- Optimal oral hygiene.

LEUKOCYTOSIS

- Abnormal increase in the number of circulating white blood cells.
- It is a reaction of the body to a pathologic situation.
- *Neutrophilia*—Occurs in acute infections, both localized and systemic, acute hemorrhage, polycythemia, myelocytic leukemia.
- *Eosinophilia*—Allergic disorders, parasitic infestation, skin diseases called pemphigus.
- *Basophilia*—Chronic myelocytic leukemia, chronic anemia, infections - chicken pox.
- *Lymphocytosis*—Acute infections like infectious mononucleosis, chronic infections like TB, syphilis, leukemia.
- *Monocytosis*—Granulomatous diseases like sarcoidosis, tuberculosis, protozoal diseases, leukemia.

INFECTIOUS MONONUCLEOSIS

(Glandular Fever) / Kissing Disease

- Clinical syndrome consisting of fever, pharyngitis and adenopathy.
- Cause : EBV virus (Epstein-Barr virus).

Transmission

- Through oropharyngeal secretions.

Clinical Features

- Fever, chills, sore throat.
- Cervical lymphadenopathy.
- Splenomegaly.
- Acute gingivitis and stomatitis.
- Palatal petechiae is the early manifestation.
- Soft palate shows edema.
- Atypical lymphocytes in blood.
- Increased antibodies to EBV.
- Positive Paul-Bunnell test—increased in heterophile antibodies (1: 4096) [i.e. antibodies against sheep RBC'S].
- Positive Monospot test, i.e. agglutination of horse RBC'S on exposure to EB virus heterophil antibodies.
- Lymphocytosis, i.e. > 50% lymphocytosis of which 10 percent are atypical.
- Thrombocytopenia.
- ESR is increased.

Treatment

- Antibiotics
- Short-term steroid therapy
- Subsides 2-4 weeks.

LEUKEMIA

It is a malignant transformation of one of the stem cell which proliferates in bone marrow and overflows into peripheral blood. May be due to overproduction of white blood cells which usually appear in the circulating blood in an immature form.

Classification

According to histogenesis

- **Myeloid (Myelogenous, myelocytic):** Involving the granulocyte series.
- **Lymphoid (Lymphogenous, lymphocytic):** Involving lymphocytic series

According to clinical courses

- Acute
- Chronic

Etiology

- Environmental factors like pesticides, viruses, ionizing radiation
- Genetic factors like Philadelphia chromosome – Translocation of Chromosomal material between 22 to 9 chromosomes in CML

Clinical Features

Age Incidence

- Acute myeloid leukemia (AML) involves broad age range including children.
- Chronic myeloid leukemia (CML) are seen in 3rd and 4th decade
- Acute lymphoblastic" (ALL) seen in children
- Chronic lymphocytic "(CLL) is the most common type and affects adults (elderly)

Clinical Features

- Malignant proliferation of stem cells displaces normal hematopoietic cells and there will be reduction in the number of WBC, RBC and platelets.
 - Fatigue, tiredness, dyspnea on exertion (due to anemia) are the common features.
 - Splenomegaly, hepatomegaly and lymphadenopathy can be seen.
- Easy bruising, petechial hemorrhages of posterior hard and soft palate.
- Gingival hemorrhage (due to thrombocytopenia)
- Fever with infection (Septicemia)
- Oral mucosal ulceration which is deep and punched out lesions in gingiva
- Oral candidiasis. Herpetic infections. Boggy, non-tender swelling of gingiva.

Diagnosis

- Immature cells in bone marrow and peripheral blood (i.e. poorly differentiated leukemic cells in bone marrow and peripheral blood).

Treatment

- Chemotherapy
- Bone marrow transplantation
- Transfusion with platelets, packed red blood cells.

DISEASES OF PLATELETS

Manifested as excessive bleeding which may result from

- Increase fragility of vessels
- Platelet deficiency or dysfunction
- Derangements in coagulation mechanism
- Combination of these.

Tests used to evaluate patients with bleeding disorders

- Skin puncture to stop bleeding
Normal value: 2-9 minutes
Bleeding time is abnormal when there is defects in platelets number or function
- Platelet count
Normal value is $150 - 450 \times 10^3/\text{mm}^3$
- Prothrombin Time (P.T)
It tests the adequacy of common and extrinsic coagulation pathway.
Prolonged P.T is seen when there is a deficiency of factor V, VII, X, prothrombin or fibrinogen.
- Partial Thromboplastin Time (PTT)
Tests the integrity of intrinsic and common clotting pathway.
Prolonged PTT is due to deficiency of V, VIII, IX, X, XI, or XII, prothrombin, fibrinogen.

Purpura:

Purplish discoloration of skin and mucous membrane due to spontaneous extravasation of blood.

Petechiae:

Pin point hemorrhagic lesions

Ecchymosis:

Extravasation of a large quantity of blood. With even larger amounts, of extravasated blood, a hematoma forms.

Hemat (blood); oma.tumor

NONTHROMBOCYTOPENIC PURPURA

- Heterogeneous group of diseases, characterized by Purpura, petechial, due to abnormalities in the blood vessel wall.

Causes

- Infections causing microbiologic damage to microvasculature
- Drug reactions
- Scurvy causes impaired collagen support of vessel wall
- Ehlers- Danlos syndrome causes impaired collagen support of vessel wall
- Hereditary hemorrhagic telangiectasia

Disorders of Platelets

Quantitative

1. Thrombocytopenia
 - a. Idiopathic thrombocytopenic purpura
 - b. Thrombotic thrombocytopenic purpura

Qualitative

- Thrombocyasthenia
- a. Familial thrombocasthenia
 - b. Thrombocytopathic purpura

THROMBOCYTOPENIA

- Thrombocytopenia, abnormal reduction in the number of circulating blood platelets.

Types

- Primary
- Secondary

Causes

- Decreased platelet production due to
 - a. Drugs and chemicals
 - b. Leukemia, aplastic anemia
 - c. Infections

- Decreased platelet survival
 - a. Infections
 - b. Drugs like quinidine, sulfa drugs
 - c. Increased Platelet sequestration – in Splenomegaly/Hypersplenism
- Due to dilution of platelets by transfusion of platelets – poor blood.

Clinical Features

- Acute form occurs in children following a viral infection
- Chronic form seen in women of child bearing age
- Profuse gingival hemorrhage
- Petechial hemorrhage on palate

Lab Findings

Platelets count < 60,000 / cu.mm

Bleeding time is prolonged

Clotting time is normal

THROMBOTIC THROMBOCYTOPENIC PURPURA

- It is immunologically mediated
- Young adults are affected
- Fever, thrombocytopenia, hemolytic anemia is seen.
- Formation of microthrombi (Composed of aggregates of platelets) in arterioles, veins and capillaries.
- Gingival tissue biopsy is highly diagnostic of this condition – occlusive subintimal deposits of PAS (periods acid Schiff) – positive material at arteriocapillary junctions.

WISKOTT-ALDRICH SYNDROME

- Hereditary disease
- Occurs in infants childhood

Clinical Features

- Thrombocytopenic purpura, petechiae, eczema
- Increase susceptibility to infections
- Decrease levels of serum IgM

Lab Findings

- Qualitative and quantitative defect in platelets
- Prolonged B.T – due to thrombocytopenia
- Platelets – alteration in size, and cell membrane

THROMBOCYTASTHENIA

Diseases characterized by a qualitative defect in blood platelets.

- **Familial Thrombocytopenia** – Hereditary. There is a membrane defect in platelets. Platelet count normal. Bleeding time is prolonged.
- **Thrombocytopenic purpura** – Unknown etiology platelets count – normal. Bleeding time – prolonged.

Clinical Features

- Both these diseases are characterized by
 - a. Gingival bleeding
 - b. Petechial hemorrhages
 - c. Excessive and prolonged bleeding from dental extractions.

THROMBOCYTHEMIA

- Increase in number of circulating blood platelets
- Prolonged bleeding despite increase in platelets count
- Though there are more number of platelets they start forming platelet thrombi leading to bleeding secondarily.

Treatment

- Radioactive phosphorus (P32)
- Blood transfusion
- Heparin, steroids – during thrombotic episodes

DISEASES INVOLVING CLOTTING FACTORS

HEMOPHILIA

- Hereditary disease where there is a defective coagulation.

Types

- Hemophilia A – Deficiency of factor VIII (Most common type)
- Hemophilia B - Deficiency of factor IX (Christmas disease)
- Hemophilia C - Deficiency of factor XI

Clinical Features

- Spontaneous hemorrhages into internal organs, joints causing massive hematoma
- Easy bruising
- Massive and prolonged gingival hemorrhage
- “Pseudotumors” in mandible -> subperiosteal bleeding with reactive bone formation causing tumor – like expansion of bone.

Lab Findings

- Prolonged coagulation time.
- Prolonged partial thromboplastin time.

Treatment

- Administration of antihemophilic factor concentrate prior to a surgical procedure.

VON WILLEBRAND'S DISEASE

- Most common inherited disorders of bleeding in humans due to deficiency of von willebrand's factor.
- vWF – (von Willebrand's factor): is a protein that forms a complex with factor VIII and circulates as a factor VIII – vWF complex.
- Important function of vWF – helps in adhesion of platelets to sub endothelial collagen -> thereby helping in homeostasis
- Serves as a carrier of factor VIII

Types

- Type 1 and type 3 – associated with a reduced quantity of circulating vWF.
- Type 2 – Qualitative defects in vWF.

Clinical Features

- Spontaneous bleeding from nose, skin, gingival
- Prolonged bleeding after extraction.

Lab Findings

- Low levels of vWF.
- Prolonged bleeding time
- Clotting time – slightly prolonged
- Prolonged partial thromboplastin time
- Absence or a decrease in the level of fibrinogen.
- Eight congenital or acquired
- Prolonged bleeding episodes
- Bleeding time – normal or slightly prolonged
- Clotting time and prothrombin time – infinite.

CRYOGLOBULINEMIA

Presence of cryoglobulins in varying amounts in blood. Cryoglobulins precipitate upon exposure to cold and redissolve upon return to body temperature.

Associated with diseases like rheumatoid arthritis, malignant lymphomas. Rarely spontaneous bleeding from the nose and mouth occurs.

DYSFIBRINOGENEMIA

- Congenital disease
- Fibrinogen is present in normal amounts but is defective in structure and coagulability.

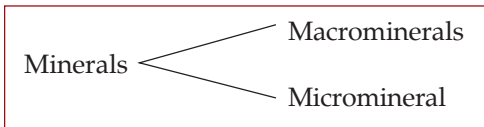
GYOGLOBULINEMIA

- Presence of Gyoglobulins in varying amounts in blood
- Gyoglobulins – precipitate upon exposure to cold and redissolve upon return to body temperature.
- Associated with diseases like rheumatoid arthritis, malignant lymphomas
- Rarely, spontaneous bleeding from the nose and mouth.

Oral Aspects of Metabolic Disease

METABOLISM

- Sum of tissue activity considered in terms of physio-chemical changes associated with and regulated by the availability, utilization, disposal of protein, fat, carbohydrate, vitamins, minerals, water and the influence of endocrines on these.



BASIC STRUCTURE OF BONE AND TEETH

CALCIUM

- 99%—In Bones.
- Serum calcium —9-11 mg/dl.
- Low serum calcium—Increases parathyroid hormone.
- Vitamin D—Increases absorption of calcium from intestine.
- Calcitonin—Opposes action of parathyroid.

HYPOCALCEMIA

- Causes tetany, carpopedal spasm, laryngospasm and convulsions.

HYPERCALCEMIA

- Causes decreased nerve conduction, muscle rigidity is seen.

HYPOPARATHYROIDISM

Decreased calcium level.

Clinical Features

- **Chvostek's sign**—Tapping of the facial muscle zygomatic process → twitching of the upper lip (Suggestive of latent tetany)
- Hypoparathyroidism during odontogenesis causes—Pitting or enamel hypoplasia, failure of tooth eruption.
- Chronic candidiasis

HYPERPARATHYROIDISM



Triad of "stones, bones and abdominal groans".

Stones

- Renal calculi, calcification of blood vessels.

Bones

- Loss of lamina dura, blurring of trabecular pattern—"Ground glass" appearance.
- Osteoporosis with attempted bone formation, new bone resorps → showing "pseudocysts".
- Pathologic fractures occurs.

"Brown Tumor"

- Tumor-reddish brown due to hemorrhage.

Radiological Features

- Unilocular/multilocular radiolucencies in mandible, ribs, clavicle.

OSTEITIS FIBROSA CYSTICA

- Central degeneration and fibrosis of long standing “brown tumor”.
- Enlargement of jaws.
- Abdominal groans: Duodenal ulcers.

Histologic Features

- Similar to “Central giant cell granuloma”.
- Vascular granulation tissue.
- Multinucleated osteoclast type giant cells.

Lab Test

- Serum calcium increased, increased alkaline phosphatase.

MAGNESIUM

- Decreased Mg → Increased parathyroid hormone.
- Increased Mg → “Tetany” identical to hypocalcemia.

PATHOLOGIC CALCIFICATION

- Abnormal deposition of calcium salts and small amounts of Mg, iron and other mineral salts
- Dystrophic
- Metastatic
- Calcinosis.

(a) Dystrophic Calcification

- Calcium salts deposited in dead or degenerating tissues.

Etiology

- Change in the local condition of the tissues (Alkalinity).

In oral cavity it is seen in:

- Gingiva, tongue, buccal mucosa, fibromas, pulp stones.

(b) Metastatic Calcification

- Precipitation of calcium salts in previously undamaged tissues.
- Excess of blood calcium causes hyperparathyroidism, hyper-vitaminosis D.

(c) Calcinosis

- Calcifications in or under the skin.
- Circumscripta -circumscribed form.
- Universalis - associated with scleroderma.

IRON

- Iron overloaded in one. Idiopathic hemochromatosis
→ excessive iron absorption “Bronze diabetes : Cirrhosis with marked brown pigmentation, diabetes, skin pigmentation.

COBALT

- Deficiency → macrocytic anemia.

FLUORIDE

- Trace quantities in human tooth, hardens enamel; cariostatic effect.
- Increased **fluoride** → osteosclerosis, collagen synthesis is affected.

DEFECTS IN PROTEIN METABOLISM

KWASHIORKOR OR MARASMUS

- Loss of tongue papillae.
- Angular cheilosis.
- Dry mouth, epithelium detached from underlying tissues.
- Retarded eruption.
- Increased incidence of dental caries, growth of jaws retarded.
- Gingiva, periodontal membrane—degeneration.

AMYLOIDOSIS

Heterogenous group of conditions characterized by the deposition of an extracellular proteinaceous substance called amyloid.

Types of Amyloid

Type A (Secondary)

- Fibrillar protein, unknown origin, deposited in genetic diseases, prolonged inflammatory diseases, familial and mediterranean fever.

Type B (Primary)

- Immune origin; seen in patients with multiple myeloma.

Type C

- Amyloid of aging, in pheochromocytoma.
Rheumatoid arthritis, multiple myeloma are predisposing factors.

Organs Involved

- Kidneys, heart, liver, spleen.

Tongue Shows Macroglossia and also Involves Gingiva

PORPHYRIA

Error in porphyrin metabolism leads to increased uroporphyrin.

Types

- Erythropoietic porphyria (congenital).
- Hepatic porphyria.

Teeth

- Brownish/ red discoloration of deciduous and permanent teeth
(Due to affinity to CaPO_4).
- Under UV light, teeth exhibit red fluorescence.

DISTURBANCE IN CARBOHYDRATE METABOLISM

- Genetically determined
- Mucopolysaccharide metabolism disturbed
→ mucopolysaccharidases.

HURLER SYNDROME

(*Mucopolysaccharidosis; Gargoylism*)

- Deficiency of D-glucuronidase.
- Intracellular accumulation of dermatan sulphate and heparan sulphate.

Clinical Features

- Large head.
- Facial features - Saddle nose, thick lips, large tongue, open mouth, prominent forehead.
- Progressive corneal clouding.
- Hepatosplenomegaly.
- Dwarfed, mentally retarded.

Oral Manifestations

- Macroglossia.
- Gingival hyperplasia.
- Broad mandible.
- Numerous impacted teeth.
- Hyperplastic dental follicles.

Histologic Features

- “Gargoyle”/ Hurler’s Cells—*Fibroblasts* with abnormal deposits of **Mucopolysaccharidosis(MPS)** [Found in gingiva].
- Large cells, crescent shaped nuclei—Stain with Alcian blue.

Lab Findings

- Increased urinary levels of MPS.
- “Reilly bodies”—Granules in circulating lymphocytes.

LIPID PROTEINOSIS

- Autosomal recessive disease.
- Waxy material deposited in submucosal connective tissue and dermis.

Clinical Features

- Laryngeal mucosa, vocal cords—First affected.

Oral Findings

- Tongue, buccal mucosa, labial mucosa - Nodular, diffusely enlarged.
- Tongue papillae destroyed.
- Recurrent painful parotitis.
- Enamel hypoplasia.
- Congenital absence of teeth.

DISTURBANCE IN LIPID METABOLISM

LIPID RETICULOENDOTHELIOSIS/LIPID STORAGE DISEASES

- Inherited disorders, deficiency of enzymes that process lipids → lipids accumulate in cells.
- **Gaucher's:** Lack of glucocerebrosidase → accumulation of glucosylceramide in macrophages.
"Lipid - laden macrophages (Gaucher's cells) = in bone marrow → Anemia, thrombocytopenia.
- Splenomegaly, hepatomegaly.
- In Mandible - ill defined radiolucencies seen.
- **Niemann Pick:** Lack of sphingomyelinase accumulation of sphingomyelin.
Histological Features : "Gaucher's cells".
"Sea Blue histiocyte" - in Neimannpick (Bone marrow aspiration).
- **Histiocytosis X Disease** (Non Lipid Reticuloendotheliosis).
- Spectrum of clinicopathologic disorders characterized by proliferation of histiocyte like cells, eosinophils, lymphocytes, plasma cells and multinucleated giant cells.

- Hand-Schuller-Christian Disease (Multifocal Eosinophilic Granuloma).
- Letterer-Siwe disease
- Eosinophilic granuloma.

HAND - SCHULLER-CHRISTIAN : DISEASE

- Skeletal, extraskeletal lesions before 5 years of age.
- **TRIAD** - “Punched out” bone destruction in skull unilateral or bilateral exophthalmous, diabetes insipidus.

Oral Manifestations

- Gingivitis, precocious exfoliation of teeth, ulcerative/proliferative mucosal lesions “scooped out” appearance in posterior mandible due to alveolar bone destruction → simulate periodontitis.
- Teeth appear “floating in air”.

Histologic Features

Four Phases

- *Proliferative histiocytic phase* : Sheets of histiocytes + eosinophils.
- *Vascular granulomatous phase*: Cholesterol laden macrophages, with histiocytes + eosinophils.
- *Xanthomatous* : Foam cells - abundant.
- Healing phase.

EOSINOPHILIC GRANULOMA

- Histiocytic proliferation with abundant eosinophils.
- Bone lesions—skull, mandible.
- Older children and adults.

Radiological Features

- Irregular parallel areas.
- Jaw lesions—resemble cysts, periapical granulomas.

LETTERER-SIWE DISEASE

- Acute, fulminating histiocytic disorders.
- Infants less than three years are involved.
- Skin, visceral organs, bone marrow are involved.
- Skin - rash (erythematous, purpuric, ecchymotic).
- Hepatosplenomegaly, lymphadenopathy, GIT, lungs.
- Diffuse skeletal involvement.

Oral Manifestations

- Ulcers, gingival hyperplasia. Premature exfoliation of teeth.

AVITAMINOSIS

Vitamin A

- Keratinizing metaplasia of epithelial cells.
- Enamel matrix—defective retarded eruption in experimental animals.

B Complex

- Riboflavin (Vit B2)
 - Angular cheilitis.
 - Atrophy - filiform papillae; fungiform papillae - engorged.
 - Tongue - coarsely granular, red.
 - Later stages - complete atrophy of papillae ® glazed, smooth magenta in color.
- Niacin
 - Stomatitis, glossitis, “bald tongue of sandwich”.
 - Oral mucosa - fiery red and painful.
 - Ulceration in interdental papillae spreads superimposed ANUG.
- Pyridoxine
 - Chelitis, Glossitis.

Vitamin C

- “Scurvy”.
- Generalized gingival swelling (boggy, violaceous).
- Spontaneous hemorrhage.
- Ulceration.

- Tooth mobility.
- Periodontal bone loss.
- Foul odour.

Teeth Changes

- Atrophy and disorganization of odontoblasts → Irregular dentin with few tubules.
- Predentin hypercalcified → heavy basophilic staining line between dentin and pulp.

Bone Changes

- Osteoblasts fail to form osteoid.

Vitamin D

Vit D deficient-rickets

- Delayed eruption of teeth.
- Developmental defects in enamel and dentin.
- Wide predentin zone, and increased interglobular dentin.

Osteomalacia

- Severe periodontitis

Vitamin D - Resistant Rickets : Familial Hypophosphatemia

- Decreased renal disorption of PO_4 and decreased intestinal calcium absorption.
- Large pulp chambers, pulp horns upto dentinoenamel junction.
→ pulp exposures, periapical abscesses, gingival sinuses.
- Microclefts in enamel.

Radiological Features

Abnormal cementum, absence of lamina dura.

HYPOPHOSPHATASIA

- Metabolic bone disease,
- Deficiency of alkaline phosphatase
- Bone abnormalities similar to rickets.

Types

- Perinatal
- Infantile
- Childhood
- Adult.

Oral Manifestations

- Presenting sign—Premature loss of primary teeth due to lack of cementum.
- Enlarged pulp chambers.
- Alveolar bone loss.

Lab diagnosis

- Increased alkaline phosphatase.
- Increased phosphoethanolamine in urine and blood.

Histologic Features

- Absence/marked reduction of cementum.

Vitamin K

- Deficiency causes gingival bleeding.

DISTURBANCES IN HORMONE METABOLISM

HYPOPITUITARISM (DWARFISM)

- Eruption and shedding of teeth - delayed.
- Small dental arch → malocclusion.
- Roots - shorter, Mandible - growth affected .
- Decreased size of teeth.

GIGANTISM

- Lips enlarged, macroglossia.
- Mandible - large.
- Acromegaly.
- Soft tissue affected → “Coarse facial features”.
- Mandibular prognathism → Apertognathia (open bite).

Hypothyroidism

(Cretinism Myxedema)

- Lips thickened due to a accumulation of GAG's
- Macroglossia
- Maxilla - over developed
- Delayed eruption, retained deciduous teeth.

HYPERTHYROIDISM

- Precocious eruption of permanent teeth.
- Early shedding of deciduous teeth.

ADDISON'S DISEASE

- Decreased production of corticosteroid. Diffuse patchy brown pigmentation.

DIABETES MELLITUS

- Periodontal disease—Progresses rapidly multiple periodontal abscesses.
- Nontender bilateral enlargement of parotid glands.
- Enlargement and erythema of attached gingiva.
- Oral candidiasis, zygomycosis.
- Xerostomia.

Progeria

(Hutchinson-Gilford Syndrome)

- Dwarfism and premature senility.
- Genetic
- Alopecia, loss of subcutaneous fat.
- Hypoplastic mandible.
- Muscle atrophy.
- Accelerated secondary dentin formation.
- Delayed eruption.

Diseases of the Nerves and Muscles

Definition

Pain

It is defined as an unpleasant sensation and emotional experience associated with actual or potential tissue damage or described in terms of such damage.

Neuralgia

- Pain in the distribution of nerves, e.g. Trigeminal neuralgia, Sphenopalatine neuralgia, Glossopharyngeal neuralgia.

Neuropathy

- Disturbance of function or pathologic change in a nerve.

TRIGEMINAL NEURALGIA

(Tic douloureux, ("painful jerking") Fothergill's disease, Trifacial neuralgia)

- Disease involving nerves which supply teeth, jaws, face and associated structures.

Etiology

- Often idiopathic.
- Occasionally brainstem tumor / infraction.
- Circulatory insufficiency.
- Cochlear artery looped against fifth nerve root near brainstem.
- Periodontal diseases, traumatic occlusion

Clinical Features

- Seen in 35 years older adults.
- Females predilection is more comparative to male.
- Right side of the face is more affected than left side.
- Maxilla and mandible both are affected.
- Pain onset is abrupt in nature due to "Trigger zone".
- Pain is lancinating type, paroxysmal and last for few seconds to few minutes.

Trigger Zones

- Vermilion border of lip
- Alae of nose
- Periorbital skin
- Cheeks
- Intraoral alveolus
- Asymptomatic refractory period is seen in between painful attacks.

Histologic Features

- Trigger points show fibrosis and infiltration of chronic inflammatory cells and myelin degeneration in gasserian ganglion.

Differential Diagnosis

- Migrainous neuralgia (pain will be persistent for few hours and no trigger zone).
- Post herpetic neuralgia (pain involves ophthalmic division and history of skin lesions).
- Sinusitis

TRIGEMINAL NEURITIS

Etiology

- Dental surgical procedures.
- Tumors of head and neck.
- Intracranial pathology.
- Pressure of a denture on dental nerves.

Clinical Features

- Pain last for hours, days and weeks.

Treatment

Medical

- Phenytoin, carbamazepine, gabapentin.
- Injection of alcohol peripherally or centrally into gasserian ganglion.

Surgical

- Peripheral neurectomy.
- Microsurgical decompression.
- Microvascular decompression (repositioning blood vessels impinging on fifth nerve).

SPHENOPALATINE NEURALGIA

(Horton's syndrome, cluster headache, periodic migrainous neuralgia, histamine cephalgia)

Etiology

- Vasodilatation of internal maxillary artery due to
 - Head trauma.
 - Abnormal release of histamine.
- Influence of hypothalamic hormone

Clinical Features

- Seen in third to fourth decade.
- Male > Female. Male predilection is more comparative to female.
- Pain is unilateral.

- Pain is radiated to mid or upper face behind the orbit, radiating to temporal or check region (zygoma), maxilla, base of nose, mastoid.
- Pain is paroxysmal (abrupt onset), intense, lancinating type.
- Pain lasts 15 min to several hours.
- No trigger zone.
- “Cluster headache” - headache occurs in clusters extended periods of remission between attacks.
- “Alarm clock” pain occurs at the same time of the day (mid night) nasal discharge, ephiphora, congestion of conjunctival blood vessels.

Treatment

- Prednisone, ergotamine
- In mild cases no treatment is needed, spontaneous resolution occurs.
- Severing the auriculotemporal nerve is done.

BELL’S PALSY

- Self limiting unilateral facial paralysis.

Triggering Events

- Acute otitis media.
- Atmospheric pressure change.
- Exposure to cold.
- Ischemia of nerve near stylomastoid foramen.
- Viral, bacterial and fungal infection.
- Multiple sclerosis.
- Pregnancy (3rd trimester).

Suspected Causes

- Reactivation of herpes simplex or zoster later in geniculate ganglion.
- Nerve demyelination.
- Ischemia or nerve edema.
- Autoimmune damage to nerves.
- Vasospasm of vessels of associated nerves.

Clinical Features

- Seen in middle age.
- Female>Male. Female predilection is more comparative to male.
- Loss of muscular control shows “mask-like appearance”.
- Inability to smile, close eyes and raise eyebrow.
- Drooping of corner of mouth causes saliva drooling.
- Abnormal taste.
- Conjunctival dryness is seen.

Treatment

- Regresses in 1 to 2 months.
- Corticosteroids therapy.
- Administration of histamine.
- Surgical decompression of nerve.
- Artificial tears and ocular antibiotic is administered.

GLOSSOPHARYNGEAL NEURALGIA

Etiology

- Ischemia.

Clinical Features

- Seen in middle or old age people.
- Sharp, shooting pain in ear, pharynx, nasopharynx and tonsil.
- Pain is short duration (30-60 secs).
- Unilateral, paroxysms of pain.
- “Trigger zone” - tonsillar fossa and oropharynx.
- Swallowing, talking, yawning/coughing triggers the condition.

Treatment

- Carbamazepine, baclofen, phenytoin.

FREY’S SYNDROME

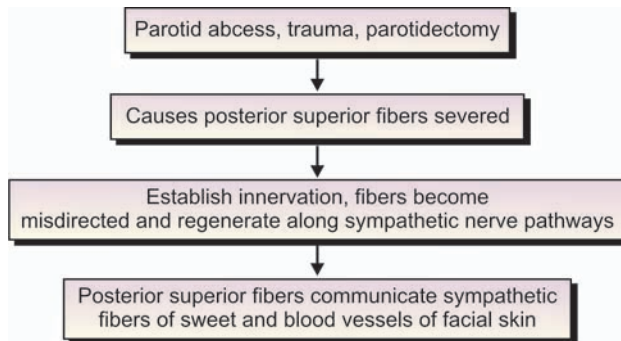
(Auriculotemporal syndrome, gustatory sweating)

- First described by Baillarger in 1953.
- Facial flushing and sweating along the distribution of auriculotemporal nerve in response to gustatory stimuli.

Supply of Auriculotemporal Nerve

- Sensory fibers to preauricular and temporal regions.
- Posterior superior fibers to parotid gland.
- Sympathetic fibers (vasomotor and pseudomotor), fibers to preauricular skin region.

Pathogenesis



Clinical Features

- Sweating
- Flushing
- Warmth and occasionally pain in the preauricular and temporal regions.

OROLINGUAL PARESTHESIA

(Glossodynia, glossopyrosis)

- It is a symptom and not a disease.

Etiology

- Anemia and pellagra.
- Diabetes.
- Hypo or hyperacidity (gastric disturbances).
- Psychogenic factors.
- Fifth nerve neuralgia.
- Xerostomia.

Clinical Features

- Glossodynia - pain in tongue
- Glossopyrosis - burning in tongue.] Tongue - common site
- Female predilection is more comparative to male (post menopausal).
- Appearance of tissues is normal.

Treatment

Analgesics, anesthetics, vitamins, antifungal.

MOTOR NEURON DISEASE

(Amyotrophies)

- Group of diseases with unknown etiology.
- Three clinically variant forms are seen.
- Progressive muscular atrophy.
- Amyotrophies lateral sclerosis.
- Progressive bulbar palsy.
- Exhibit tongue, pharyngeal, laryngeal muscle involvement or limb muscle involvement.

PROGRESSIVE MUSCULAR ATROPHY

- Seen in childhood.
- Progressive weakness of limbs causes difficulty in walking and leg pain can be seen.

AMYOTROPHIC LATERAL SCLEROSIS

Clinical Features

- Seen in 40-50 years of age.
- Limb weakness, difficulty in swallowing.
- Indistinct speech and hoarseness of voice.
- Atrophy of tongue.

PROGRESSIVE BULBAR PALSY

- Atrophy of face, masseter and temporal muscles, tongue is commonly seen.

- Difficulty in mastication and swallowing occurs.
- Impairment of palate and vocal cords also occurs.

MULTIPLE SCLEROSIS

(Demyelinating disease)

Etiology

- Allergic manifestations of the nervous tissue due to antigen - antibody reactions.
- Venous thrombosis in nervous system leads to this condition.
- Repeated vasoconstriction in various portions of nervous system may cause multiple sclerosis.

Clinical Features

- Seen in 20 to 40 years.
- Visual disturbances like nystagmus, diplopia.
- Fatigability, weakness and stiffness of extremities.

Charcot's Triad

- Intention tremor
- Nystagmus
- Dysarthria or scanning speech.
- Imperfect speech articulation.
- Staccato type of speech—"interrupted speech".
- Bell's palsy, fifth nerve neuralgia—in some patients.

OROFACIAL DYSKINESIA

Etiology

- Extrapyrarnidal disorders.
- Complication of phenothiazine therapy.

Clinical Features

- Seen in above 60 years of age.
- Rarely denture wearers.
- Severe involuntary movements of facial, oral, (tongue, lips) and cervical musculature.

Treatment

- Surgical.
- Anti-Parkinsonian drug therapy.

MENTERE'S DISEASE

- Etiology is unknown
- Manifests as deafness, tinnitus, and vertigo.

MIGRAINE

(Severe periodic headache)

Etiology

- Vasoconstriction of cerebral arteries (portions) leads to pre-migraine aura and causes vasodilatation, especially external carotid artery.

Clinical Features

- Seen in second decade
 "Preheadache phenomenon"
- Hallucinations
- Scotomas scintillations
- Vertigo
- Aphasia
- Unilateral paresthesia or facial weakness
 "Headache phase"
- Unilateral - temporal, frontal, retro-orbital throbbing pain
- Vomiting.

Treatment

- Administration of aspirin, codeine, norepinephrine, ergotamine.

TEMPORAL ARTERITIS

- Focal granulomatous inflammation of temporal artery.
- Immunologic reaction to elastic lamina of artery.

Clinical Features

- Female > male → Female predilection is more comparative to male.
- Headache, throbbing pain in teeth and TMJ, scalp and occiput.
- Increased ESR and leukocytosis.
- Eyepain, photophobia, diplopia.
- Muscle aching and stiffness follows an acute condition called polymyalgia rheumatica.

Treatment

- Corticosteroids.

CAUSALGIA

- Severe pain which arises after injury to sectioning of a peripheral sensory nerve.

Etiology

- After extraction of multi-rooted tooth (traumatic).
- Pain is burning type.
- Triggering factors—touch, heat, cold, emotional disturbance.

Treatment

- Resection of nerves.

ATYPICAL FACIAL PAIN

- Vague, deep, poorly localized pain in regions of V, IX, II and III cranial nerves.
- Pain lacks trigger zone, crosses midline.
- Persists for weeks to months.
- Any facial pain for which a definitive diagnosis is not possible.

Etiology

- Common after tonsillectomy.

Differential Diagnosis

- Eagle's syndrome.

- Elongation of styloid process/ossification of stylohyoid ligament.
- Dysphagia, otalgia, headache, vague orofacial and pharyngeal pain.

DISEASES OF MUSCLES

DYSTROPHY

- Degeneration of muscle fibres leads to severe generalized familial muscular dystrophy and generalized muscle atrophy.
- Inability to walk.

Cause

- Postural defects in muscles of mastication, facial, laryngeal and pharyngeal muscles.

Clinical Features

- Mild restricted muscular dystrophy.
- First involves muscles of shoulders and face.
- Lips protrude - "taper lips".
- Inability to smile.

MYOTONIA

- Failure of muscle relaxation after cessation of voluntary contraction, alterations in facial muscles.

Dystrophic Myotonia

- Atrophy of masseter causes.
- Narrowing of lower half of the face.
- Ptosis.
- Myopathic facies.
- Pharyngeal, laryngeal muscles exhibit weakness.

Congenital Myotonia

- Myotonia and hypertrophy (Increase in size of cells).
- Masseter, muscles of neck shows hypertrophy.

Acquired Myotonia

- Spasm of muscles occurs clonuses (spasm is intermittent) trismus (constant spasm).

Causes

- Epilepsy
- Tetanus
- Local infections—third molar infection.

Paramyotonia

- Nonprogressive myotonia not associated with muscular wasting.
- Stiffness of muscles occurs on face and tongue on exposure to cold.

Hypotonias

- Complete absence of tonus in muscle, leads to muscle weakness.

MYASTHENIA GRAVIS

- Progressive weakness of skeletal muscle following activity.
- Defect in neuromuscular transmission.

Clinical Features

- Seen in adults.
- Muscles of mastication and facial expression are involved.
- Difficulty in deglutition and mastication.
- Taste disturbances.

Treatment

- Physostigmine (acetyl choline esterase).

MYOSITIS

- Inflammation of muscle tissue.

Causes

- Infections
- Physical
- Chemical
- Dermatomyositis
- Myositis ossificans—Generalized traumatic
- Proliferative myositis
- Focal myositis.

Types

- Acute form shows skin eruptions and calcification occurs. Nodules are seen, leads to calcinosis cutis, calcinosis universalis.
- Chronic form has no skin involvement; muscle stiffness and weakness.

Oral Manifestations

- Stomatitis
- Pharyngitis
- Muscles of jaws
- Tongue pharynx involved.

GENERALIZED MYOSITIS OSSIFICANS

- Masses of bone and fibrous tissue in muscles shows atrophy.

Clinical Features

- Nodular swellings is seen in neck and back.
- Masseter muscle ossifies and jaw is fixed.

TRAUMATIC MYOSITIS OSSIFICANS

- Ossification in muscle following trauma.
- “Riders bone” -Seen in thigh muscles of jockeys.

Oral Manifestations

- Involves masseter and temporal muscles causes difficulty in mouth opening called **Proliferative myositis**.

- Pseudosarcomatous process of muscle proliferation of basophilic giant cells and fibroblasts involving perimysium, epimysium and neighboring fascia.

Etiology

- Mechanical trauma.

Clinical Features

- Firm nodule.

Atrophy

- Decrease in size of muscle fibers.

Hypertrophy

- Increase in size of muscle fibers.

DEFINITION

- Biopsy is the removal of tissue from the living organism for the purpose of microscopic examination and diagnosis.

Types of Biopsy

- Excisional biopsy
- Incisional biopsy
- Fine needle aspiration cytology (FNAC)
- Frozen section biopsy.

Excisional Biopsy

- If a lesion is totally excised for histological evaluation, it is called excisional biopsy. It is done in small lesions.

Incisional Biopsy

- When only a small section of tissue is removed from the lesion for the purpose of histological evaluation, it is called incisional biopsy. It is done in large lesions.

Fine Needle Aspiration Cytology (FNAC)

- It is done by aspirating tissue material from inside a lesion, which is later on diagnosed microscopically after preparing a smear. It is performed in glandular or cystic lesion.

Frozen Section Biopsy

- It is performed in order to get an immediate histological report of a lesion. The tissue obtained is kept deep freeze and the frozen tissue is sectioned and stained to get diagnosis.

EXFOLIATIVE CYTOLOGY

- It is a microscopic study of cells obtained from the surface of an organ or lesion after suitable staining. The neoplastic cells are less cohesive than other normal cells usually they shed on surface of the lesion. The shed neoplastic cells are obtained from lesion by scrapping into surface and are then evaluated for possible changes like dysplasia or malignancy.

Technique of Exfoliative Cytology

- First the surface of lesion is cleaned by removing all the debris and mucus.
- Gentle scrapping is done on surface of lesion with moistened tongue blade.
- The materials present on surface of lesion are collected at border of instrument.
- The collected material is then evenly spread over a microscopic slide and immediately fixed with either 95 percent alcohol or equal parts of alcohol and ether.
- The slide is then air-dried and is stained by special stain called papanicolaou stain.

Indications of Exfoliative Cytology

- Herpes simplex
- Herpes zoster
- Pemphigus vulgaris
- Squamous cell carcinoma

- Aphthous ulcer
The findings in exfoliative cytology is categorized as.

Classes

Class I

- Normal
- The findings indicate that only normal cells are present in smear.

Class II

- Atypical
- The findings indicate presence of minor cellular atypia, but no evidence of malignancy.

Class III

- Intermediate
- The cells display wider atypia that may be suggestive of cancer, but the features are not clear cut.

Class IV

- Suggestive of cancer
- The lesion indicates that there is preserve of few cells with malignant characteristics. Biopsy is mandatory.

Class V

- Positive of cancer.
- The cells exhibit definite features of malignancy, biopsy is mandatory.

Healing of Oral Wound

- Healing of wounds is the most interesting phenomenon which characterizes a living organism. The ability of damaged tissue to repair itself is a response of life itself and within this very process may lie the final understanding of nature. It is said that an unhealed wound will eventually result in the death of an organism healing of wound as a complex series of biological events.
- Healing of tissue is generally considered to be a phase of the inflammatory reaction, since it cannot be separated from the proceeding vascular and cellular phenomena occurring in response to and injury.
- Healing on the other hand is the body response to injury in an attempt to restore normal structure and function. The process of healing involves two distinct processes:
 - Regeneration
 - Repair

FACTORS AFFECTING HEALING OF ORAL WOUNDS

Age

- Faster in young individual due to higher rate of tissue metabolism.

Types of Tissue

- Different types of tissue exhibit a great deal of variation in healing potential, e.g. epithelial tissue heals faster than neural tissue.

Location of Wound

- Wound located near vascular area heals faster than area with least blood supply.

Mobility of Wound

- If the wound site is subjected to movement, the rate of wound healing is delayed.

PHYSICAL FACTORS AFFECTING THE HEALING

Trauma

- Rate of wound healing is affected by continuous trauma at the life of injury.

Local Temperature

- If local temperature in an area is high and well maintained, then healing process occurs at faster rate.

Radiation

- Low dose of radiation stimulates healing process.

Circulatory Factors

- Very good blood supply heals faster.

Infections

- Low-grade infection stimulate healing.

Hormonal Factors

- Trephones (wound hormones) released by proteolytic breakdown of cellular debris accelerate healing process.

HEALING OF BIOPSY WOUND

Healing by First Intention (Primary Healing)

- When the cut surfaces of biopsy wound is approximated or closely sutured, the wound heals up by primary intension.
- In the initial phase, there will be formation of blood clot, which helps to hold the parts of wound together.
- The tissue becomes edematous and an inflammatory process starts, with the infiltration of polymorphonuclear neutrophils (PMN) and lymphocytes into the area.
- The tissue debris collected in wound are cleared either by process of phagocytosis or by their lysis with the help of proteolytic enzymes, liberated by inflammatory cells.
- Once the tissue debris are cleared, granulation tissue forms and that replaces the blood clot in the wound, it equally consists of young blood capillaries, proliferating fibroblasts, polymorph nuclear neutrophils and other leukocytes.
- The epithelium at the edge of wound starts to proliferate gradually and it covers the entire wound surface.
- Finally, the healing process is complete with progressive increase in amount of dense collagen bundles and/with decrease in number of inflammatory cells in area.

Healing by Secondary Intention

- When the opposing margins of wound can not be approximated together by suturing, the wound fills in from the base with the formation of a larger amount of granulation tissue, such type of healing of open wound is known as healing by secondary intention.
- The secondary healing occurs essentially by same process as seen in primary healing, the only difference is that a more severe inflammatory reaction and an exuberant fibroblast and endothelial cell proliferation occurs later.
- In secondary healing, once the blood clot is removed, the granulation tissue fills up the entire area and epithelium begins to grow over it, until the wound surface is completely epithelialized.
- Later on the inflammatory exudates disappear slowly and the fibroblasts produce large amounts of collagen.

- Most of the healing processes occurring due to secondary intention, result in scar formation at the healing site.

HEALING OF EXTRACTION WOUND

Healing in an extraction socket occurs as:

- Soon after tooth is extracted, bleeding occurs in the socket and the clot is formed.
- Within a day, the periphery of the clot shows edema and neutrophilic cells infiltration.
- In the next two days fibroblasts and endothelial cells proliferate into surrounding bone marrow spaces and they gradually enter into clot. This process is called organization of clot.
- At the same time, removal of debris like dead cells, necrotic tissue dead pieces of bone from the wound also takes place, and it is done by neutrophils, macrophages and osteoclast cells.
- The clot organization process is completed normally by a week following this, the epithelium at the periphery of wound starts growing and it gradually covers the entire socket area.
- Later on the inflammatory cells decrease in number and the collagen fibers increase in the area.
- In about 10-15 days, immature bone or osteoid starts forming at the margin of the socket. They move into socket and gradually fill up the entire socket by replacing the granulation tissue.
- Finally, in about 3 weeks to 6 months time, the entire socket is filled with mature bone that replaces the osteoid.

DRY SOCKET

(Alveolar osteitis)

- Dry socket is a common complication in healing of extraction wounds. It follows complicated tooth extraction and commonly occurs in mandibular premolar or molar areas.
- The dry socket is usually a very painful condition and the patient often has a foul breath.
- Clinical examination, reveals a socket devoid of clot bony walls of socket are barely visible.
- Histological sections of socket wall reveal the formation of necrotic bone, containing empty lacunae.

- There is intense inflammatory reaction in surrounding bone.
- Dry socket is a localized osteitis and not an osteomyelitis and the condition heals very slowly.
- Zinc oxide eugenol pack is often given in the socket for palliative reaction.

Appendices

APPENDIX I

CLASSIFICATION RELATED TO ORAL PATHOLOGY

(White Lesions)

(A) Variation in Structure and Appearance of Normal Mucosa

- Leukoedema.
- Fordyce's granules.
- Linea alba.

(B) White Lesion with Definite Precancerous Potential

- Leukoplakia.
- Erythroplakia.
- Tobacco keratosis, actinic keratosis.
- Lesion associate with electrogalvanism.
- Carcinoma in situ.
- Verrucous carcinoma.
- Lichen planus.
- Lichenoid reaction.
- Oral submucous fibrosis.
- Dyskeratosis congenital.
- Lupus erythematosus.
- Acanthosis nigricans.

(C) White Lesion without Precancerous Potential

- Traumatic keratosis.
- Focal epithelial hyperplasia.

- Psoriasis.
- Geographic tongue.
- Pachyonychia congenital.
- White sponge nevus.
- Hereditary benign epithelial dysplasia.
- Stomatitis nicotina.
- Hyperkeratosis palmoplantaris with gingival hyperkeratosis.
- Darier's disease.
- Intraoral skin grafts.
- Pseudoxanthoma elasticum.
- Hyalinosis cutis et mucosa oris
- Oral condyloma acuminatum
- Hairy leukoplakia

(D) Nonkeratotic White Lesion

- White hairy tongue
- Burns – thermal, chemical, electrical
- Desquamative gingivitis
- Epidermolysis bullosa
- Pemphigus
- Benign mucous membrane pemphigoid (BMMP)
- Diphtheria
- Syphilitic mucus patches
- Acute necrotizing ulcerative gingivitis (ANUG)
- Koplik's spot of measles
- Candidiasis
- Median rhomboidal glossitis
- Verruca vulgaris

PRECANCEROUS LESION AND CONDITION

(A) Precancerous Lesion

- Leukoplakia.
- Erythroplakia.
- Palatal lesion associated with reverse smoking.
- Verrucous hyperplasia.
- Carcinoma in situ.

(B) Precancerous Condition

- Oral submucous fibrosis.
- Sideropenic anemia.
- Erosive lichen planus.
- Discoid lupus erythematosus.
- Dyskeratosis congenital.

RED LESIONS OF ORAL MUCOSA

(A) Inflammatory Condition

- Inflammation associated with traumatic injury.
- Mechanical – cheek biting, ill fitting denture, sharp edge, overhanging restoration and ecchymoses due to trauma.
- Chemical – aspirin, formocresol and TCA.
- Thermal – hot food, hot beverage and hot instrumentation.
- Radiation – mucositis, cheilitis.
- Infection.
- Bacterial.
- Scarlet fever red fever.
- Gonococcal stomatitis.
- Vincent infection or gingivostomatitis.
- Acute pharyngitis, tonsillitis.
- Fungal.
- Atrophic candidiasis.
- Angular cheilitis.
- Acute pseudomembranous candidiasis.
- Viral.
- Measles – Koplik's spot.
- Lymphonodular pharyngitis.
- Herpes simplex infection.
- Herpes zoster.
- Herpangina.
- Chickenpox.
- Hand-foot-mouth disease.
- Allergic/immunological.
- Pyogenic granuloma.
- Giant cell epulis.
- Pregnancy tumor.

- Traumatic hemangioma.
- Inflammatory fibrous hyperplasia.
- Desquamative gingivitis.

(B) Congenital – Hereditary Developmental Condition

- Hemangioma
- Sturge-Weber syndrome
- Rendu-Osler-Weber disease
- Hereditary hemorrhage telangiectasia
- Median rhomboidal glossitis
- Geographic tongue
- Melkersson-Rosenthal syndrome
- Crohn's disease

(C) Vascular Disease and Blood Disorder

- Purpura.
- Polycythemia.
- Agranulocytosis.
- Polyarteritis nodosa.
- Leukemia.

(D) Dermatological

- Pemphigus.
- Erythema multiforme.
- Stevens-Johnson syndrome.
- Lichen planus.
- Lichenoid reaction.
- Epidermolysis bullosa.
- Psoriasis.
- Lupus erythematosus.

(E) Other Systemic Disease

- Uremic stomatitis.
- Diabetes stomatitis.
- Scurvy.
- Pernicious anemia.
- Ulcerative stomatitis.

(F) Premalignant and Malignant Lesion

- Atrophic leukoplakia.
- Erythroplakia.
- Squamous cell carcinoma.
- Carcinoma in situ.
- Kaposi sarcoma.
- Hemangioendothelioma.

VESICULOBULLOUS LESIONS

I. First Classification

(A) Hereditary

- Epidermolysis bullosa
- Familial benign chronic pemphigus
- Dyskeratosis congenital
- Acrodermatitis enteropathica

(B) Viral Infection

- Primary herpetic gingivostomatitis
- Secondary herpetic gingivostomatitis
- Varicella (chickenpox)
- Shingles (herpes zoster)
- Measles
- Hand, foot and mouth disease
- Smallpox and cat-scratch disease
- Infectious mononucleosis
- Herpangina
- Acute lymphnodular pharyngitis
- AIDS

(C) Mucocutaneous Diseases

- Pemphigus vulgaris
- Pemphigus vegetans
- Benign mucous membrane pemphigoid
- Bullous pemphigoid
- Lichen planus
- Toxic epidermal necrolysis

(D) Miscellaneous

- Oral submucous fibrosis
- Hyperacidity or gastritis
- Constipation and malabsorption syndrome
- Impetigo
- Oral blood blister
- Erythema multiforme

II. Second Classification

(A) Vesicular lesion

(i) Nonfebrile lesion

- Recurrent herpes labialis
- Recurrent herpes stomatitis
- Reiter's syndrome
- Contact stomatitis
- Impetigo
- Dyskeratosis congenital

(ii) Febrile lesion

- Herpetic gingivostomatitis
- Herpangina
- Hand-foot and mouth disease
- Chickenpox
- Herpes zoster
- Smallpox

(iii) Bullous lesion

- Pemphigus
- Familial benign chronic pemphigus
- Bullous pemphigoid
- Benign mucous membrane pemphigoid
- Epidermolysis bullosa
- Dermatitis herpetiformis
- Erythema multiforme
- Stevens-Johnson syndrome

CYSTS OF THE ORAL CAVITY

I. Odontogenic

(A) Developmental

- Primordial cyst.
- Odontogenic keratocyst.
- Gingival cyst of infant.
- Gingival cyst of adult.
- Lateral periodontal cyst.
- Dentigerous cyst.
- Eruption cyst.
- Calcifying epithelial odontogenic cyst.
- Chondroma

(B) Inflammatory

- Radicular cyst.
- Periodontal cyst.
- Inflammatory collateral cyst.
- Paradental cyst.

II. Nonodontogenic

- Nasopalatine cyst.
- Median palatine cyst.
- Median alveolar cyst.
- Median mandibular cyst.
- Globulomaxillary cyst.
- Nasolabial cyst.
- Nasoalveolar cyst.
- Simple bone cyst.
- Hemorrhagic bone cyst.

III. Cysts Associated with Maxillary Sinus

- Benign mucosal cyst of maxillary antrum.
- Mucocele – mucus retention cyst.
- Surgical ciliated cyst.

IV. Soft Tissue Cysts

- Dermoid
- Epidermoid
- Branchial
- Thyroglossal
- Anterior median lingual cyst
- Oral cyst with gastric epithelium
- Cystic hygroma
- Parasitic cyst, hydatid cyst, cysticercosis cellulosae

V. Cysts of the Salivary Gland

- Mucocele
- Ranula

CYSTS OF THE JAWS

(A) Epithelial

- Developmental.
- Odontogenic.
- Gingival cyst of infants.
- Odontogenic keratocyst (primordial cyst).
- Dentigerous (follicular) cyst.
- Eruption cyst.
- Lateral periodontal cyst.
- Gingival cyst of adults.
- Botryoid odontogenic cyst.
- Glandular odontogenic (sialo-odontogenic; mucoepidermoid odontogenic) cyst.
- Calcifying odontogenic cyst.
- Cysts of the oral regions.

(B) Non-odontogenic

- Nasopalatine duct (incisive canal) cyst.
- Nasolabial (nasolabial) cyst.
- Midpalatal raphe cyst of infants.
- Median palatine, median alveolar and median mandibular cysts.
- Globulomaxillary cyst.

(C) Inflammatory

- Radicular cyst, apical and lateral.
- Residual cyst.
- Paradental cyst and mandibular infected buccal cyst.
- Inflammatory collateral cyst.

(D) Non-epithelial

- Solitary bone cyst (traumatic, simple, hemorrhagic bone cyst.
- Aneurysmal bone cyst.

(E) Cysts Associated with the Maxillary Antrum

- Benign mucosal cyst of the maxillary antrum.
- Postoperative maxillary cyst (surgical ciliated cyst of the maxilla).

(F) Cysts of the Soft Tissues of the Mouth, Face and Neck

- Dermoid and epidermoid cysts.
- Lympho-epithelial (branchial cleft) cyst.
- Thyroglossal duct cyst.
- Anterior median lingual cyst (intralingual cyst of foregut origin).
- Oral cysts with gastric or intestinal epithelium (oral alimentary tract cyst).
- Cystic hygroma.
- Nasopharyngeal cysts.
- Thymic cyst.
- Cysts of the salivary glands: mucous extravasation cyst; mucous retention cyst; ranula; polycystic (dysgenetic) disease of the parotid.
- Parasitic cysts : hydatid cyst; cysticercus cellulosae; trichinosis.

CLASSIFICATION OF TUMOR

<i>Tissue of origin</i>	<i>Benign</i>	<i>Malignant</i>
Epithelial tumors		
Squamous epithelium	Squamous cell papilloma	Squamous cell carcinoma
Transitional epithelium	Transitional cell papilloma	Transitional cell carcinoma
Glandular epithelium		Adenoma adenocarcinoma
Basal cell layer		Basal cell carcinoma
Neuroectodermal	Nevus	Melanoma
Hepatocyte	Liver cell adenoma	Hepatocellular carcinoma
Mesenchyma		
Adipose tissue	Lipoma	Liposarcoma
Adult fibrous tissue	Fibroma	Fibrosarcoma
Embryonic fibrous tissue	Myxoma	Myxosarcoma
Cartilage	Chondroma	Chondrosarcoma
Bone	Osteoma	Osteosarcoma
Synovium	Benign synovioma	Synovial sarcoma
Skeletal muscle	Rhabdomyoma	Rhabdomyosarcoma
Smooth muscle	Leiomyoma	Leiomyosarcoma
Mesothelium		Mesothelioma
Blood vessels	Hemangioma	Angiosarcoma
Lymph vessels	Lymphangioma	Lymphangiosarcoma
Glomus cell	Glomus tumor	
Meninges	Meningioma	Invasive meningioma
Hemopoietic cell		Leukemia
Lymphoid tissue		Malignant lymphoma
Nerve sheath	Neurilemmoma	Neurogenic sarcoma
Nerve cell	Ganglioneuroma	Neuroblastoma
Mixed tumors		
Salivary gland	Pleomorphic adenoma	Malignant PA
<i>Tumor of more than one germ cell layer</i>		
Totipotent cells in gonads or in embryonal rests	Mature teratoma	Immature teratoma

DISORDERS OF THE TEMPOROMANDIBULAR JOINT

(A) Developmental

- Agenesis of the condyle.
- Hypoplasia of the condyle.
- Hyperplasia of the condyle.
- Exostosis of the joint.

(B) Traumatic

- Trauma to disc.
- Trauma to developing condyle or growth center.
- Trauma to ligament.
- Subluxation or dislocation or dislocation within displacement.
- Ankylosis.
- Foreign body in the joint space.
- Trauma to muscle

(C) Inflammatory

- Infective.
- Pyogenic infection from staphylococci.
- Syphilis.
- Tuberculosis.
- Actinomycosis.
- Rheumatic arthritis.
- Osteomyelitis
- Psoriasis
- Secondary to hepatitis B infection
- Non-infective
- Still's disease
- Systemic lupus erythematosus
- Ankylosis spondylitis
- Synovitis
- Non-articular arthritis
- Myositis
- Myositis ossificans
- MPDS

(D) Metabolic Disorders

- Gout
- Chondrocalcinosis
- Oxalosis
- Amyloidosis
- Wilson's disease
- Gaucher's disease

(E) Neoplasms

- Benign
- Osteoma
- Osteochondroma
- Fibromyxoma
- Synovioma
- Malignant
- Osteosarcoma
- Chondrosarcoma
- Synovial sarcoma
- Malignant lymphoma

(F) Neuropathic

- Charcoat joint
- Ritter's sympathetic dystrophy

(G) Internal Derangement

- Disc displacement
- Disc fracture

AUTOIMMUNE DISORDERS

(A) Associated with Mucocutaneous Lesion

- Recurrent aphthous ulcer
- Behcet's disease
- Pemphigus
- Bullous pemphigoid
- Cicatricial pemphigoid
- Dermatitis herpetiformis

(B) Salivary Gland

- Mikulicz's disease
- Sjogren's syndrome

(C) Blood Disorder

- Pernicious anemia
- Purpura

(D) Collagen Disorder

- Systemic lupus erythematosus
- Scleroderma
- Rheumatic arthritis

(E) Miscellaneous

- Myasthenia gravis
- Dermatomyositis
- Oral submucous fibrosis

BLOOD DISORDERS

I. Disorder of RBC

(A) Deficiency

- Iron deficiency anemia
- Pernicious anemia
- Normocytic anemia
- Aplastic anemia
- Hemolytic anemia
- Extracorporeal causes
- Infection and toxin
- Hypersplenism
- Rh factor incompatibility (hemolytic disease of newborn, erythroblastosis fetalis).
- Chronic liver disease.
- Autoimmune diseases.
- Transfusion reaction.
- Intracorporeal causes.
- Hereditary spherocytosis.

- Sickle cell anemia.
- Thalassemia.
- Cooley's anemia.
- Glucose-6 phosphate dehydrogenase deficiency.
- Pyruvate kinase deficiency.
- Folic acid and B₁₂ deficiency.

(B) Polycythemia

- Relative.
- Secondary.
- Vera (malignant).

II. Disorders of WBC

(A) Qualitative

- Lazy leukocyte syndrome.
- Chediak-Higashi syndrome.

(B) Quantitative

- Agranulocytosis.
- Cyclic neutropenia.
- Myeloblastic leukemia.
- Decrease of eosinophils.

III. Disorders of Platelet

(A) Purpura

- Idiopathic thrombocytopenic purpura.
- Secondary thrombocytopenic purpura.
- Thrombotic thrombocytopenia.
- Drug associated thrombocytopenia.
- Thrombocytopathia.
- Glanzmann's thrombasthenia.
- von Willebrand's disease.
- Bernard-Soulier syndrome.
- Aldrich syndrome.

(B) Thrombocytosis

Disorder of coagulation

- Hemophilia A.
- Hemophilia B.
- Factor XI deficiency.
- Factor X deficiency.
- von Willebrand's disease.

FIBRO-OSSEOUS LESIONS

(A) Lesions Arising from Periodontal Ligament

- Periapical cemental dysplasia
- Localized fibro-osseous cemental lesion
- Florid osseous dysplasia
- Ossifying fibroma
- Cementifying fibroma

(B) Fibrous dysplasia

- Monostotic
- Polyostotic
- Familial

(C) Fibro-osseous Neoplasm of Uncertain Origin or Debatable Origin

- Cementoblastoma
- Osteoblastoma
- Osteoid osteoma
- Juvenile ossifying fibroma
- Fibro-osteoma or osteofibroma
- Cementifying fibroma
- Paget's disease
- Ossifying fibrous epulis

(D) Giant Cell Lesion

- Central giant cell tumor or central giant cell reparative granuloma
- Brown tumor of hyperthyroidism
- Aneurysmal bone cyst
- Cherubism

ODONTOGENIC TUMORS (MODIFIED WHO CLASSIFICATION)

I. Benign

(A) Odontogenic Epithelium without Odontogenic Ectomesenchyme

- Ameloblastoma.
- Squamous odontogenic tumor.
- Calcifying epithelial odontogenic tumor (Pindborg tumor).
- Adenomatoid odontogenic tumor.

(B) Odontogenic Epithelium with Odontogenic Ectomesenchyme with or without Hard Tissue Formation

- Ameloblastic fibroma
- Ameloblastic fibrodentinoma
- Ameloblastic fibro-odontoma
- Odontoameloblastoma
- Calcifying odontogenic cyst
- Complex odontoma
- Compound odontoma

(C) Odontogenic Ectomesenchyme with or without Included Odontogenic Epithelium

- Odontogenic fibroma.
- Myxoma (myxofibroma).
- Cementoblastoma (benign cementoblastoma true cementoma).

2. Malignant

(A) Odontogenic Carcinomas

- Malignant ameloblastoma.
- Primary intraosseous carcinoma.
- Clear cell odontogenic carcinoma**.
- Ghost cell odontogenic carcinoma.

(B) Odontogenic Sarcomas

- Ameloblastic fibrosarcoma.
- Ameloblastic fibrodentinosa sarcoma.
- Ameloblastic fibro-odontosarcoma.

APPENDIX II

Normal Laboratory Values

Test	Material used	Normal comments
Albumin	Serum	3.5-6.0
Amino acid nitrogen	Serum	80-150 units
Ascorbic acid	Serum or plasma	0.7-2.0
Basal metabolic rate		Minus 15% to plus 15% (BMR)
Bilirubin	serum	Direct : 0-0.2 Total : 0.1-1.0
Bromide	Serum or plasma	Less than 50
Bromsulphalein test	Serum. Liver function test is valueless in patients with obvious jaundice	Less than 10%, retained in 30 min
Calcium, diffusible	Serum, consists of ionized and nonionized calcium	4.5-5.0
Calcium, nondiffusible	Serum, nonionized calcium, contains protein-bound calcium fraction	4.5-6.0
Calcium, total	Serum total calcium equals diffusible plus nondiffusible	9.0-11.5 (4.5-5.5 mEq/liter)
Calcium, total	Feces, 24-hour specimen	70-90% of ingested calcium eliminated in feces
Calcium, total	Urine, 24-hour specimen	10-30% of ingested calcium eliminated in urine
Carbon dioxide-combining power (CO ₂ capacity)	Serum or plasma. Normal Milli-equivalent values expressed as bicarbonate or carbonic acid	Adults : 55-70 vols% (25-35 mEq/liter) Children: 40-55 vol% (18-25 mEq/liter)
Carotenoids	Serum	80-400 µg/100 ml
Cephalin flocculation	Serum. Liver function test	Below 2+ in 48 hr
Chloride	Serum or plasma	570-620 (as NaCl) 340-370 (as Cl) 96-105 mEq/liter as Cl)
Chloride	Spinal fluid	720-750
Chloride	Urine, 24-hours specimen	10-16 gm/24 hr
Cholesterol, esters	Serum or plasma	80-200
Cholesterol, total	Serum or plasma	120-260
Congo red test	Serum or plasma. Test for Amyloidosis and Nephrosis	10-30% eliminated from Blood in 1 hr

Contd...

Contd...

<i>Test</i>	<i>Material used</i>	<i>Normal comments</i>
Copper	Serum	100-200 µg/100 ml
Creatine	Whole blood	3-7
Creatine	Urine, 24-hours specimen	Adults: 0-200/24 hr Children: 10-15/24 hr
Creatinine	Serum	0.6-1.3
Creatinine	Urine, 24 hour specimen	1-1.8 gm/24 hr
Fatty acids, total	Serum	250-500
Fibrin	Plasma	0.3-0.6 gm/100 ml
Glucose	Whole blood	60-90
	Serum	70-105
	Postprandial	Less than 140
Glucose	Spinal fluid	40-60
Hippuric acid	Urine. Liver function test	
	Oral	3 gm benzoic acid excreted/4 hr
	Intravenous	0.7-0.95 gm benzoic acid excreted/1 hr
Hydrogen ion concentration	Whole blood, serum or plasma	7.3-7.5 units
Icteric index	Serum	4-6 units
Iodine, protein-bound	Serum	3-8 micrograms/100 ml
Lecithin	Serum or plasma	225-250
Lipase	Serum	0.8-1.5 units (Alper) 0.2-1.5 units (Cherry- Crandall)
Lipids,	Total serum	470-750
Nitrogen, nonprotein	Whole blood	25-35
Nitrogen, urea	Whole blood	9-17
pH	Whole blood, Serum or plasma	7.3-7.5 units
Phenolsulfonph- thalein	Urine. Renal function test	40-60% 1st hour (PSP) 20-25% 2nd hour
Phosphatase, acid	Serum	0-2.5 units (King- Armstrong) 0-1.5 Phenol units
Phosphatase, alkaline	Serum	Adults: 1.5-5.0 unit (Bodansky) 5-10 unit (King- Armstrong) 1.0-3.5 phenol units Children : 5-14 units (Bodansky)

Contd...

Contd...

Test	Material used	Normal comments
		15-20 units (King-Armstrong)
Phospholipids	Serum	4-12 phenol units
Phosphorus (inorganic)	Serum	150-350
		Adults:
		3.0-4.5
		Children:
		4.5-6.0
Potassium	Serum	16-22 (4.1-5.6 mEq/liter)
Protein total	Serum	6-8 gm/100 ml
Albumin		3.2-4.1 gm/100 ml
Alpha globulin		0.7-1.5 gm/100 ml
Beta globulin		0.7-1.3 gm/100 ml
Gamma globulin		0.7-1.3 gm/100 ml
Total globulin		2.6-3.8 gm/100 ml
Albumin-globulin ratio (A/G ratio)		1.5-2.5 : 1
Protein, total	Spinal fluid	20-40
Sodium	Serum	315-340 (137-147 mEq/liter)
Sulfates	Serum	2.5-5.0
Thymol turbidity	Serum. Liver function test	0-4 units
Uric acid	Whole blood	2-4
Urobilinogen	Feces, 4-day specimen	150-300
Urobilinogen	Urine, 24-hour specimen	8 or less
Vitamin A	Serum	15-60 µg/ml
Vitamin C	Serum or plasma	0.7-2.0
Volume, blood	Whole blood and plasma	70-100 cc blood/kg
		35-50 cc plasma/kg
Zinc turbidity	Serum. Liver function test	2-8 units
All values expressed as milligrams per 100 ml unless otherwise specified		

Normal Red Blood Cell Values

Age	Red cell count (millions per cu mm blood)	Hemoglobin (gm per 100 ml)	Hematocrit (vol packed cells per 100 ml)	Sedimentation rate in 1 hr (Wintrobe method)	Reticulocytes (% of erythrocytes)
<i>Children:</i>					
First year	6.1-4.5	25-11.2	64-35	0-2 (at birth)	2-6 (at birth) 0.5-1.5 (2.5 days after birth)
2-10 years	4.6-4.7	11.5-12.9	35.3-37.5	3-13 mm	0.5-1.5
11-15 years	4.8	13.4	39		0.5-1.5
<i>Adult:</i>					
Females	4.2-5.4	12-16	37-47	0-15 mm	
Males	4.6-6.2	14-16	40-54	0-6.5 mm	0.5-1.5

Type of cell	Percent	Average	Absolute number	
			Minimum	Maximum
Total leukocytes	-	7000	5000	10,000
Myelocytes	0	0	0	0
Juvenile neutrophils	3-5	300	150	400
Segmented neutrophils	54-62	4000	3000	5800
Eosinophils	1-3	200	50	250
Basophils	0-0.75	25	15	50
Lymphocytes	25-33	2100	1500	3000
Monocytes	3-7	375	285	500

From MM Wintrobe : Clinical Hematology, 6th ed. Philadelphia, Lead and Febiger, 1967.

Normal Blood Platelet Values and Associated Phenomena

Total number of platelets	:	150,000-400,000 per cu mm blood
Bleeding time	:	Under 5 minutes (Duke's method)
Clotting time	:	1-7 minutes (Capillary tube method)
	:	2.5-5 minutes (Kruse and Moses method)
	:	5-10 minutes (Lee and White method)
Prothrombin time	:	10-20 seconds (Quick method)
Clot retraction time	:	Qualitative begins 1-6 hours; complete at 24 hours
	:	Quantitative 80-90%

Contd...

Contd...

Capillary fragility : More than 10 petechiae per 1 inch
(tourniquet test): circle – positive

Heterophile antibodies : Below 1:56 dilution
(Sheep cell agglutination)

Incidence of blood groups in
normal population:

Group O : 40%

A : 45

B-12

AB-4

Normal Average values of urine

Water 95% of total urine

Inorganic substance

Sodium (as Na₂O) 4.0 gm/liter

Potassium (as K₂O) 2.0 gm/liter

Calcium (as CaO) 0.2 gm/liter

Magnesium (as MgO) 0.2 gm/liter

Iron 0.003 gm/liter

Organic constituents

Urea 15.25 gm/liter

Uric acid 0.4-0.6 gm/liter

Creatinine 0.8-1.5 gm/liter

Ammonia 0.6 gm/liter

Undetermined N 0.6 gm/liter

Traces of other substances

BS 70-110 mg%

PPBS 90-160 mg%

RBS 80-120 mg%

Urea M : 15-40 mg%

F : 10-30 mg%

Creatinine : M : 0.6-1.2 mg%

F : 0.4-1.0 mg%

Cholesterol 130-250 mg%

TGL 50-170 mg%

HDL 35-75 mg%

LDL 80-180 mg%

VLDC 10-30 mg%

CHO/HDC Ratio : Less than 5.0 : 1

Calcium 8.5-11.0 mg%

Contd...

Contd...

Phosphorous			3.0-4.8 mg%
Uric acid			2.5-7.0 mg% (M)
			1.5-6.0 mg% (F)
Serum bilirubin	(T)	=	1.0 mg%
Serum bilirubin	(D)	=	0.3 mg%
Serum bilirubin	(TD)	=	0.2-0.8 mg%
SGPT			5-40 IU/L
SGDT			5-40 IU/L
Alkaline phosphate			82-306 IV/L
Total protein			6.0-8.0 gm%
Albumin			3.8-5.0 gm%
Globin			2.3- 3.5 gm%
Albumin/Globulin ratio	2:1		
Amylase upto			290 IU/L
Prostate Specific Antigen ((PSA) :			upto 4.0 mg/ml
Sodium	=		135-150 mEq/L
Potassium	=		3.5-5.0 mEq/L
Chloride	=		95-103
Bicarbonate	=		20-28
HbA1C			
Good condition			7.0-8.0 %
Poor condition			8.0-9.0 %
Thyroid function test			
		T ₃	82-180 mg/dl
		T ₄	4.5-11.5 µg/dl
		TSH	0.3-4.0 IU/ml
		FT3	2.3-4.2 Pg/ml
		FT4	0.8-2.0 mg/dl

APPENDIX III

I. Microscopic Diagnostic Terms

- **Acantholysis** Dissolution of the intercellular bridges of the prickle cell layer of the epithelium.
- **Agranulocytosis** A marked decrease in the number of granulocytes, particularly neutrophils.
- **Anaplastic** Pertaining to adult cells that have changed irreversibly toward more primitive cell types. Such changes are often malignant.
- **Atypical** Irregular, not conformable to the type.
- **Benign** Not malignant; neoplasms that do not threaten life.
- **B lymphocyte** A lymphocyte, also called a B-cell, that matures without passing through the thymus. It matures into plasma cells that produce antibodies.
- **Capsule** Compressed fibrous connective tissue around a benign neoplasm separating it from surrounding tissues.
- **Carcinoma** A malignant growth made up of epithelial cells that are capable of infiltration and metastasis.
- **Central** In oral pathology, a lesion occurring within bone.
- **Chronic** Persisting over a long time; when applied to a disease, chronic means that there has been little change or extremely slow progression over a long period.
- **Cyst** A pathologic epithelium-lined cavity, usually containing fluid or semisolid.
- **Differentiation** Development of specialized shape and/or function; acquisition of individual characteristics.
- **Diffuse Growth Pattern** A growth pattern exhibited by a lesion that is not well demarcated.
- **Degeneration** Reversible pathologic changes with cells.
- **Dysplasia** An abnormality of development characterized by the loss of normal cellular architecture.
- **Epithelium** The cellular makeup of skin and mucous membranes.
- **Erosion** A denudation of epithelium above the basal cell layer.
- **Etiology** The study or theory of the factors that cause disease and their introduction to the host.
- **Exudate** Material that has escaped from blood vessels into tissue or onto the surface of a tissue, usually because of inflammation.

- **Granuloma** A tumor-like mass of inflammatory tissue consisting of a central collection of macrophages, often with multinucleated giant cells, surrounded by lymphocytes.
- **Granulomatous** Pertaining to a well-defined area that has developed as a reaction to the presence of living organisms or a foreign body. The tissue consist primarily of histiocytes.
- **Histiocyte** A large phagocytic cell from the reticuloendothelial system. The reticuloendothelial system is a network made up of all of the phagocytic cells in the body, which included macrophages, Kupffer cells in the liver, and microglia of the brain.
- **Hyperchromatic** Staining more intensely than normal.
- **Hyperorthokeratosis** Keratin is the outermost layer of epithelium as seen under the microscope and is seen in two forms: orthokeratin and parakeratin. Orthokeratin has no visible nuclei within the outer layer, whereas in parakeratin nuclei are present. Hyperorthokeratosis is the presence of excess orthokeratin.
- **Hyperplasia** An increase in the size of a tissue or organ due to an increase in the number of constituent cells.
- **Hypochromic** Stained less intensely than normal.
- **Hypoplasia** Incomplete development of a tissue or organ; a tissue reduced in size because of a decreased number of constituent cells.
- **Invasion** The infiltration and active destruction of surrounding tissues.
- **Keratinization** The formation of microscopic fibrils of keratin in the keratinocytes (keratin-forming cells). In the oral cavity the term is used to describe changes in the outer layer of the epithelium.
- **Lymphocyte** A variety of leukocyte or white blood cell that is important to the immune response and that arises in the lymph nodes. Lymphocytes can be large or small, and are round, nongranular, and classified as either T- or B-lymphocytes.
- **Lymphoid tissue** Tissue composed of lymphocytes supported by a meshwork of connective tissue.
- **Macrophage** A large, mononuclear phagocyte derived from monocytes. Macrophages become mobile when stimulated by inflammation and interact with lymphocytes in an immune response.

- **Malignant** A neoplastic growth that is not usually encapsulated, grows rapidly, and can readily metastasize.
- **Malignant tumor** Cancer; a tumor that is resistant to treatment and frequently causes death; a tumor that has the potential for uncontrolled growth and dissemination or recurrence, or both.
- **Margination** A phenomenon that occurs during the relatively early phases of inflammation in which white blood cells tend to occupy the periphery of the blood vessels and adhere to endothelial cells that line the vessels.
- **Mesenchymal** The meshwork of embryonic connective tissue in the mesoderm that gives rise to the connective tissue of the body, blood vessels, and lymph vessels.
- **Mitotic figure** Dividing cells caught in the process of mitosis.
- **Monocyte** A mononuclear phagocytic leukocyte with an ovoid or kidney-shaped nucleus, containing lacey, linear chromatin, and abundant gray-blue cytoplasm filled with fine, reddish and azurophilic granules.
- **Necrosis** The death of a cell as a result of injury or disease.
- **Neoplasia** Characterized by the presence of new and uncontrolled cellular growth.
- **Neoplasm** A mass of newly formed tissue; a tumor.
- **Neutrophil** A medium-sized white blood cell with a nucleus consisting of three to five lobes and a cytoplasm containing small granules; one of a group of white blood cells called granulocytes, the others being eosinophils and basophils. Neutrophils make up about 65 percent of the white blood cells in normal blood. Also known as polymorphonuclear leukocyte, PMN, or “poly”.
- **Pathogenesis** The process by which an etiologic agent produces a disease.
- **Phagocytosis** A process of ingestion and digestion by cells.
- **Platelet** One of the elements found in circulating blood. A platelet has a circular or disk-like shape is small; hence the term platelet. Platelets aid in blood coagulation and clot retraction.
- **Pleomorphic** Occurs in various forms.
- **Proliferation** The multiplication of cells.
- **T lymphocyte** A lymphocyte that passes through the thymus before migrating to tissues. The T-lymphocyte, also called a T- cell, is responsible for cell-mediated immunity.
- **Ulcer** Loss of surface tissue due to a sloughing of necrotic

inflammatory tissue. The defect extends into the underlying lamina propria.

II. Radiographic Terms Used to Describe Bony Lesions

- **Radiopaque** A light area on a radiograph indicating presence of a material that impedes passage of X-rays.
- **Radiolucent** A dark area on a radiograph indicating presence of a material that does not impede passage of X-rays.
- **Mixed radiolucent and radiopaque** A radiographic lesion composed of a mixture of radiolucencies and radiopacities; indicates a mixture of soft and hard tissues.
- **Unilocular** A term used to describe a radiographic appearance of a single, rounded compartment or locule that is well defined or outlined.
- **Multilocular** A term used to describe a radiographic appearance of a lesion that extends beyond the confines of one distinct area and is defined as multiple, rounded compartments of locules or parts that are somewhat fused together, making up the entire lesion. These can appear “soap bubble-like” or “honeycomb-like”.
- **Diffuse** In the description of a lesion, borders of the lesion are not well defined, and it is not possible to detect the exact parameters of the lesion.
- **Localized** Confined to a limited part, not general or systemic.
- **Coalescing** The process by which parts of a whole join together, or fuse, to make one.
- **Expansile** Capable of being extended or expanded.
- **Well circumscribed** Term used to describe a lesion whose borders are specifically defined, and in which one can clearly see the exact margins and extent; well defined (well delineated) border.
- **Scalloped appearance** A radiolucent lesion that extends between the roots; fluted border.
- **Root resorption** Breakdown or destruction of root structure; loss of root structure. The apex of the tooth appears shortened or blunted and irregularly shaped.
- **Ground glass** Fine radiopaque spots in radiolucent background
- **Cotton wool** Confluent radiopacities
- **Punched out radiolucency** Small areas of radiolucency

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